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THE ASSOCIATION BETWEEN THE SO-CALLED PERINUCLEAR BASOPHILIC GRANULES AND THE ELIMINATION OF THE ALLOXURIC BODIES IN THE URINE.

(From the Medical Clinic of Prof. Kraus in Graz.)

By T. B. Futcher, M. B., *Instructor in Medicine, Johns Hopkins University and Asst. Res. Physician, Johns Hopkins Hospital.*

In 1894 Neusser described a peculiar granulation in the leucocytes of patients suffering from a uratic diathesis. This term was given a wide meaning, and under it he included gout, uratic lithiasis, as well as the various forms of "irregular gout," as muscular rheumatism, nervous asthma, skin affections, gastro-intestinal derangements, diabetes, leukæmia, neuralgia and neurasthenia. These granules were brought out by staining freshly dried specimens of the blood with a modified Ehrlich's triacid mixture in which the basic ingredient was relatively increased. They are basic staining granules, and with this mixture appear as greenish black or dense black droplets over and about the nuclei of the leucocytes. They vary considerably in size, the smallest being about the size of the neutrophilic granules, and the largest considerably larger than the eosinophilic granules. Often they have a glistening or refractile appearance. They are always in immediate contact with the nucleus, never being present amongst the ordinary granules of the leucocytes. They give one the idea that they constitute some substance which has been squeezed out of the nucleus. Neusser found them most abundant in the mononuclear leucocytes, in which they often form a complete ring about the nucleus, but stated that they were also present in the polynuclear leucocytes and eosinophiles. He believed that they were of the nature of a nucleo-albumin in composition, and saw in their presence a sign for an increased uric acid production in the organism. This assumption was based on the analysis of the urine of 100 patients, in whom, along with the already described blood condition, an elimination of from 0.8 to 1.5 grams of uric acid, and a uric acid coefficient within the limits of 1:30 to 1:20 (1:50 being normal) were found. It is important to note that the patients on whom these observations were made were not brought under a condition of nitrogenous equilibrium.

The clinical interest of these basophilic granules would be very great if it could be proven that in an extended series of cases they were associated only with a uric acid diathesis and were entirely absent in other affections. If such could be proven we would then have a ready clinical means of differentiating symptoms due to a uratic diathesis from those arising from some other cause. Neusser himself states that they are also to be found in a certain percentage of cases of tuberculosis, and believes them to be of prognostic value. He claims to have

found that cases showing the granules run a more favorable course, and that the lung infiltration is more likely to undergo fibroid change than in cases where the granules are absent.

Kolisch, a pupil of Neusser's, has advanced the theory that a uric acid diathesis is not due to an anomaly of in the formation of or in the relative solubility of uric acid, but in the increased production of the alloxuric bodies (uric acid + xanthin bases), out of the products of nuclein destruction. He found that in cases where the perinuclear granules were abundant that there was a definite increase in the quantity of alloxuric bodies eliminated. The increase was due to a marked increase of the xanthin bases, the uric acid being relatively diminished; and it was to the presence of these bases circulating in the blood that the symptoms of a uratic diathesis were due, and not to any anomaly in the formation or excretion of uric acid. Neusser and Kolisch, although they both believe that the basophilic granules bear an intimate association with the causation of the uratic diathesis, differ in their views as to which ingredient of the urine is increased by their presence. Neusser found an increased elimination of uric acid, while Kolisch found that the uric acid was relatively diminished, and the xanthin bases markedly increased, resulting in a total increase in the amount of the alloxuric bodies eliminated.

With the exception of the above difference, Neusser and Kolisch agree that the occurrence of the perinuclear basophilic granules indicates an increase in the nuclein constituents of the blood. Direct analyses of the blood for the amount of nuclein contained in it were naturally not made; and pure color reactions, even from a qualitative standpoint, are unreliable, as shown by L. Heine. If one accept without further questioning Neusser's view that the perinuclear granulation is a morphological criterium for an over-production of nuclein derivatives from the cell nuclei, it is not easily explainable why an increase in the quantity of the alloxuric bodies eliminated in the urine should follow. If one considers these granules identical with the pyrenogenic granules of Löwit found in the leucocytes of the river crab (in the arthropoda the uric acid is not formed from the nuclein materials) and in certain normal leucocytes of the bone-marrow, then they fall under the general heading of karyorhexis or breaking up of the nucleus, and would more likely indicate a chromatolytic degeneration of the leucocytes. Prof. Kraus was able to demonstrate similar granules in the liver cells when portions of the liver substance were removed from the body while still warm and kept in a moist chamber at 40° C. At a definite stage in the breaking down of the nuclei he found granules in the protoplasm of the liver cells resembling and staining similarly to those found in the leucocytes by Neusser. If such a condition takes place under any circumstances, either physiological or pathological, in the living person, an increase in the alloxuric bodies eliminated might be expected.

In studying the subject of Neusser's granules I have endeavored to determine whether there is any regular coincidence between the presence of these granules and the elimination of the alloxuric bodies, and further, whether the granules are found only in cases showing the symptoms of a uratic diathesis.

Doubt as to this intimate association arose in my own mind while examining the blood of patients for the basophilic granules, at first without studying their effect on metabolism. In a very large number of cases examined, both in healthy and diseased persons, I have never failed to find the granules present. Four of these cases were cases of true gout with typical joint affections and well defined tophi in the ears. In two of these cases the granules were very abundant, but not more so in the mononuclear than in the polymorphonuclear leucocytes. In the remaining two cases they were on the whole not very abundant, and the polymorphonuclears showed the granules more numerous than did the mononuclears. Further, I was able to convince myself, both in Baltimore and in Graz, that the granules were not more marked in the blood of patients suffering from the so-called uratic diathesis than in other diseases, or even in certain apparently healthy individuals. The fact that the granules are found in other diseases than in uratic diathesis, and also in healthy persons, lessens their clinical interest. Neusser believed their presence in tuberculosis was a favorable sign. In this disease I have observed cases with abundant granules and others with few granules without noticing any difference in its subsequent course. The granules vary in richness in the leucocytes from day to day in the same individual, notwithstanding his living under the same conditions of nourishment, etc. This fact seems to diminish the significance of the granules. It seems more common for variations of this kind to occur than for the granules to remain constant from day to day. No method seemed practicable for making an accurate count of these granules, and in the following cases reported it seemed sufficient for all practical purposes to stain the specimens of dried blood and to compare from day to day the amount of granulation present in the different forms of leucocytes.

The following cases, in which the basophilic granules and their effect on metabolism were studied, were undertaken especially to ascertain whether the amount of granulation present really influenced the quantity of the alloxuric bodies eliminated in the urine.

In all, 8 cases were carefully studied. Blood specimens were stained each day with Neusser's staining mixture, and the same technique followed from day to day. The patients were as nearly as possible brought under the conditions of nitrogenous equilibrium before the observations on the urine were concluded. Each case was followed for a period of 5 to 6 days, and during this time the same amount of food was taken each day, and the body weight from day to day remained practically constant. The total amount of nitrogen ingested in the food and eliminated in the urine and fæces was estimated daily. In all the cases the alloxuric bodies were estimated, and in two cases the uric acid as well. The nitrogen was determined according to the method of Kjeldahl, the uric acid according to Ludwig's, and the alloxuric bodies according to the Krüger-Wulf* methods.

In order to assist in the understanding of the following tables I give what is considered the normal amount of nitrogen in grams contained in the alloxuric bodies for the 24 hours, as found by various observers who have made determinations up to the present date.

*Zeit. für physiol. Chemie, Bd. XX.

For the 24 hours:

Kolisch gives	0.260 gram.
Weintraud	(1) 0.344–0.360
	(2) 0.433–0.534
Richter	0.380
Magnus–Levy	0.506
Richter	0.387
I myself found in healthy persons	(1) 0.499
	(2) 0.551

Judging from these analyses it may be considered that values above 0.4 gram are physiologically high, while those above 0.5 gram may be regarded as pathological.

Case I.—Dr. K., aged 32 years, weight 79.5 kilograms, with a well-marked tendency to a gradually increasing corpulence. Not inclined to undertake great muscular exertion, but in every respect perfectly healthy. After nitrogenous equilibrium had been established, 1.5 liters of Carlsbad water (Mühlbrunn) were drank daily for a period of 8 days.

The daily diet was as follows: Ham, 150 grams; roast beef, 100 grams; milk, 500 grams; 6 breakfast rolls; butter, 65 grams; rice, 60 grams; sugar, 3 pieces; black coffee (infusion), 100 grams; sherry, 100 grams; soda water, 2 bottles.

The above diet represented about 1968 calories (about 25 calories per kilogram body weight), which were made up as follows:

Albumin	362.17 calories.
Fat	837.73
Carbohydrates	646.35
Alcohol	122.15
Total	1968.40

TABLE I.—THE URINE.

Date.	Day of observation.	Daily amount of urine in ccm.	Total nitrogen in grams.	Nitrogen in alloxuric bodies in grams.	Uric acid—nitrogen in grams.	Percentage of alloxuric body—nitrogen of the total nitrogen.
July 6	1	995	14.7638	0.6042	.....	4.16
" 7	2	1240	12.8898	0.4318	.....	3.35
" 8	3	1630	14.7759	0.5262	.....	3.56
" 9	4	1200	14.0700	0.4169	.....	2.97
" 10	5	1080	15.5547	0.4430	.....	2.84
" 11	6	1160	15.0666	0.5602	0.2550	3.71
" 12	7	1630	15.4947	0.5058	.....	3.26
" 13*	8	2270	15.7582	0.4149	.....	2.63
" 14	9	2800	14.1159	0.4459	.....	3.16
" 15	10	2345	15.3560	0.4042	.....	2.63
" 16	11	2700	13.9482	0.3780	.....	2.71
" 17	12	3120	15.5719	0.3849	.....	2.47
" 18	13	3045	12.9062	0.3810	.....	2.95
" 19	14	2920	14.9825	0.3244	.....	2.16
" 20	15	2840	14.3732	0.4025	0.2613	2.80

*From 13 to 20 Carlsbad water was drunk.

TABLE II.—NITROGEN BALANCE (N IN GRAMS).

Date.	Day of observation.	Body weight in kilograms.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Feces.		
July 10	5	79.300	.....	15.5547		.....	.....
" 11	6	79.800	16.2347	15.0666	1.1832	16.2498	.....
" 12	7	79.900	15.9769	15.4947	1.1832	16 6779	—0.7010
" 19	14	79.050	.....	14.9825	.....	.....	.....
" 20	15	79.050	18.6598	14.3732	3.3325	17.7057	+0.9541

Condition of the leucocytes with reference to Neusser's granules: During the period in which no Carlsbad water was taken the granules were present only in very moderate numbers. On the other hand, from July 16th to 20th, during which the water was drank, there was a very distinct increase in the number of granules present in all the forms, particularly in the mononuclear leucocytes. At the same time it will be seen that there was a marked diminution in the alloxuric bodies as represented by the amount of nitrogen eliminated.

Observation II.—Dr. L., a perfectly healthy man, aged 25 and weighing 69.5 kilograms. Muscular, moderate panniculus adiposus, and of a quiet disposition. My colleague, after bringing himself to a point of equal daily nitrogen elimination, drank Carlsbad water for a period of 4 days (1.5 liter of Mühlbrunn per day).

The daily diet was as follows: Ham, 250 grams; veal, 250 grams; milk, 100 grams; wine, 500 grams; tea (black infusion), 250 grams; rum, 15 grams; butter, 50 grams; breakfast rolls, 3.

The above food represented about 1895 calories daily (27 calories per kilo body weight), which were made up as follows:

Albumin	511.73 calories.
Fat	785.49
Carbohydrates	341.69
Alcohol	256.88
Total	1895.79

TABLE III.—URINE.

Date.	Day of observation.	Daily amount of urine in ccm.	Total nitrogen in grams.	Nitrogen in alloxuric bodies in grams.	Uric acid—nitrogen in grams.	Percentage of alloxuric body—N of total nitrogen.
June 12	1	1430	25.2421	0.5710	.....	2.27
" 13	2	1815	30.1310	0.5595	.....	1.85
" 14	3	1800	28.1925	0.5237	.....	1.85
" 15	4	1880	30.3996	0.5791	.....	1.90
" 16	5	2000	30.0650	0.5217	0.4222	1.73
" 17*	6	1890	27.2868	0.6515	.....	2.38
" 18	7	3110	29.4983	0.5421	.....	1.83
" 19	8	2840	28.40	0.6908	0.4222	2 43
" 20	9	2730	27.30	0.6162	.....	2.10

*From June 17 to 20 Carlsbad water was drunk.

TABLE IV.—NITROGEN BALANCE (N IN GRAMS).

Date.	Day of observation.	Body weight in kilograms.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Feces.		
June 14	3	69.700	.....	.....	.....	.....	.....
" 15	4	69.700	31.7345	30.3996	.....	.....	.....
" 16	5	69.800	31.5645	30 0650	1.3142	31.3792	+0 8536
" 19	8	70.200	31.6679	28.40	.....	.....	.....
" 20	9	70.000	31.5979	29.30	1.2619	30.5702	+1.5643

Relation of Neusser's granules in the leucocytes: In the period before Carlsbad water was taken, with the amount of the alloxuric bodies above the normal (and with a large amount of uric acid), the granules were not particularly abundant. In the second half (June 17–20), when the water was taken, the amount of the alloxuric bodies eliminated was still higher,

whereas the granules had become distinctly diminished in number, particularly in the small mononuclears.

Observation III.—A. F., a young woman, 33 years old, and a cook by occupation. She had a severe anæmia (red corpuscles 1,900,000 per ccm., leucocytes 6800, and hæmoglobin 25 per cent, according to Fleischl's hæmometer). The temperature was normal during the observation. Pulse averaged 110 per min. Slight dyspnœa even when at rest. Patient was of rather large frame, and the panniculus adiposus was not particularly reduced. No œdema; urine free from albumin. Had suffered previously from some gastric trouble, but the symptoms had disappeared. From June 25th to July 7th her weight had become reduced from 57 to 53.4 kilograms.

The daily diet was as follows: Ham, 100 grams; white bread, 100 grams; milk, 500 grams; wine (white), 250 grams; water, about 1000 grams.

The diet administered represented about 928 calories (17 per kilo body weight), which were made up as follows:

Albumin	207.46 calories.
Fat	219.20
Carbohydrates	379.49
Alcohol	122.50
Total	928.65

TABLE V.—URINE.

Date.	Day of observation.	Daily amount in ccm.	Spec. weight.	Total nitrogen in grams.	Nitrogen in alloxuric bodies in grams.	Percentage of alloxuric body—N of the total nitrogen.
July 1	1	1300	1022	.....	.....	.....
" 2	2	1400	1010	10.0940	0.4827	4.78
" 3	3	1100	1014	8.8358	0.4832	5.46
" 4	4	950	1018	8.0964	0.3608	4.44
" 5	5	1100	1016	9.9715	0.3735	3.72
" 6	6	750	1028	7.7963	0.3793	4.86

TABLE VI.—NITROGEN INGESTED (IN GRAMS).

Date.	Day of observation.	Ham.	White bread.	Milk.	Wine.	Total nitrogen.
July 1	1	4.4480	1.5236	2.9925	0.0568	9.0209
" 2	2	4.4480	1.5236	2.9925	0.0568	9.0209
" 3	3	4.4480	1.5236	2.9225	0.0568	8.9509
" 4	4	4.4480	1.5236	2.9225	0.0568	8.9509
" 5	5	4.4480	1.5236	2.9575	0.0568	8.9859
" 6	6	4.4480	1.5236	2.9575	0.0568	8.9859

TABLE VII.—NITROGEN BALANCE (N IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
July 1	1	54.200	9.0209	.....	.....	.....	.....
" 2	2	54.300	9 0209	10.0940	0.3879	10.4819	—1.4610
" 3	3	54.200	8.9509	8.8358	0.3879	9.2237	—0.2728
" 4	4	53 900	8.9509	8.0964	0.3879	9.4843	—0.5333
" 5	5	53.600	8.9859	9.9715	0.3879	10.3594	—1.3735
" 6	6	53.200	8.9859	7.7963	0.3879	8.1842	+0.8017

Relation of Neusser's granules in the leucocytes: On the day before the analyses were begun the granules were present in very small numbers in the leucocytes. This was also the case on July 1st and 2nd, when a relatively high amount of alloxuric bodies was being eliminated. On July 3rd there was an apparent and on the following days a very marked increase in the number of granules, whilst the nitrogen of the alloxuric bodies both absolutely and in percentage became diminished.

Observation IV.—Patient was a woman 45 years of age. Fourteen years ago she had her first severe attack of articular rheumatism, although she has had milder attacks since 1881. Both hands at present show characteristic deformities. Since 1891 she has had symptoms of some cardiac lesion. In 1895, tricuspid and aortic insufficiency was diagnosed. During the period that the patient was under observation she was comparatively well; good diuresis and no œdema. Average pulse-rate, 80; respirations, 22. Moderate body exertion was possible without dyspnœa. Cyanosis was quite marked.

The diet was as follows: Ham, 100 grams; breakfast rolls (4), 208 grams; milk, 500 grams; wine (white), 250 grams; tea (infusion), 500 grams; rum, 30 grams; sugar (6 pieces), 30 grams; Rohitsch water, 300 grams.

These food materials represented about 1286 calories (26 calories per kilo body weight), which were made up as follows:

Albumin	234.19 calories.
Fat	223.47
Carbohydrates	643.19
Alcohol	185.50
Total	1286.35

TABLE VIII.—URINE.

Date.	Day of observation.	Daily amount of urine in grams.	Spec. gravity.	Total nitrogen in grams.	Nitrogen of alloxuric bodies in grams.	Percentage of alloxuric body—N of the total nitrogen.
July 19	1	2400	1012	8.2320	0.3276	3.97
" 20	2	2800	1007	9.6530	0.4459	4.61
" 21	3	2150	1012	9.9706	0.3517	3.52
" 22	4	1400	1014	9.6530	0.3463	3.58
" 23	5	700	1023	9.9356	0.4383	4.41

TABLE IX.—NITROGEN INGESTED (IN GRAMS).

Date.	Day of observation.	Ham.	Bread.	Milk.	Wine.	Tea and rum.	Total nitrogen.
July 19	1	5.2052	3.3473	2.9837	0.0785	0.1312	11.7459
" 20	2	5.2052	3.3473	2.9837	0.0785	0.1312	11.7459
" 21	3	5.2052	3.3473	2.8787	0.0785	0.1312	11.6409
" 22	4	5.2052	3.3473	2.6950	0.0785	0.1312	11.4572
" 23	5	5.2052	3.3473	2.9312	0.0785	0.1312	11.6934

TABLE X.—NITROGEN BALANCE (N IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
July 19	1	47.700	11.7459	8.2320	0.9985	9.2305	+2.5158
" 20	2	47.400	11.7459	9.6530	0.9985	10.6515	+1.0944
" 21	3	46.800	11.6400	9.9706	0.9985	10.9691	+0.6718
" 22	4	45.700	11.4572	9.6530	0.9985	10.6488	+0.8084
" 23	5	46.000	11.6934	9.9356	0.9985	10.9341	+0.7593

Relation of Neusser's granules in the leucocytes: On the first two days of the observation the granules were compara-

tively few. On July 21 they were relatively increased, whilst the alloxuric bodies, which had been comparatively high on the 20th, were distinctly diminished in quantity.

Observation V.—The patient was a brewer, 30 years old, with hypertrophic cirrhosis of the liver. Since 1895 he has had icterus, with tenderness in the region of the liver. The jaundice has had a tendency to disappear and reappear. The fæces at times would be distinctly bile-tinged, and at other times free from biliary coloring matter. Appetite good. At present there is a characteristic enlargement of the liver and spleen, no ascites, and no well marked evidence of collateral circulation. No elevation of temperature. Rather poorly nourished; weight has varied between 62 and 59 kilograms since April 10. Has a retinitis. Urine is free from albumin.

The daily diet was as follows: Ham, 300 grams; bread, 700 grams; wine, 500 grams; tea (infusion), 500 grams; rum, 30 grams; sugar (6 pieces), 30 grams; soda water, 4 bottles.

These food materials represent about 2806 calories (47 calories per kilo body weight), which were arranged as follows:

Albumin	516.27 calories.
Fat	256.22
Carbohydrates	1726.10
Alcohol	308.00
Total	2806.58

TABLE XI.—URINE.

Date.	Day of observation.	Daily amount.	Specific gravity.	Total nitrogen in grams.	Nitrogen of alloxuric bodies in grams.	Percentage of alloxuric body—N of total nitrogen.
May 15	1	1630	1026	13.2511	0.4773	3.60
" 16	2	1830	1023	14.6564	0.4153	2.83
" 17	3	1550	1023	16.2778	0.5089	3.12
" 18	4	1700	1024	17.2238	0.4918	2.85
" 19	5	2040	1024	20.2051	0.4118	2.03
" 20	6	1600	1022	16.4352	0.4771	2.90

TABLE XII.—NITROGEN INGESTED (IN GRAMS).

Date.	Day of observation.	Ham.	Bread.	White wine.	Total nitrogen.
May 15–19	1–5	13.0454	7.9398	0.2625	21.2477
" 20	6	11.3237	7.9398	0.2625	19.5260

TABLE XIII.—NITROGEN BALANCE (N IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
May 15	1	59.200	21.2477	13.2510	2.2526	15.5036	+5.7741
" 16	2	58.500	21.2477	14.6564	2.2526	16.9090	+4.3387
" 17	3	58.500	21.2477	16.2778	2.2526	18.5304	+2.7173
" 18	4	58.900	21.2477	17.2238	2.2526	19.4764	+1 7713
" 19	5	59.400	21.2477	20.2051	2.2526	22.4577	−0 7900
" 20	6	60.000	19.5260	16.4352	2 2526	18.6878	+0.3611

Relation of Neusser's granules in the leucocytes: Several days before the commencement of the chemical analyses the granules were much more numerous than they were during the period of examination. There was no variation observed during the latter period. Altogether the granules were only moderately numerous, but not more so in the mononuclear than in the other varieties of leucocytes. Some of the mononuclears were without granules. It will be seen that the amount of the alloxuric bodies was relatively high.

Observation VI.—This patient was a man also with hypertrophic cirrhosis of the liver. Since 1893 he had complained of gastric and intestinal symptoms. In 1895 enlargement of the liver and spleen was noticed. Icterus has been persistent, with the exception of one interval when he was free. Weighed 49 kilo when he came to the clinic, but has gained slightly since. The blood examination showed 2,500,000 red corpuscles, 7900 leucocytes, and 55 per cent hæmoglobin (Fleischl).

The daily diet was as follows: Ham, 300 grams; white bread, 450 grams; milk, 500 grams; wine (red), 300 grams; tea (infusion), 500 grams; rum, 30 grams; sugar (6 pieces), 30 grams; Rohitsch water, 2 bottles.

The above diet represented about 2370 calories (48 per kilo body weight), which were made up as follows:

Albumin	515.65 calories.
Fat	385.02
Carbohydrates	1161.38
Alcohol	308.00
Total	2370.05

TABLE XIV.—URINE.

Date.	Day of observation.	Daily amount of urine in ccm.	Specific gravity.	Total nitrogen in grams	Alloxuric body—N in grams.	Percentage of alloxuric body—N of the total nitrogen.
June 7	1	2500	1015	14.4813	0.6650	4.59
" 8	2	2050	1017	16.3590	0.6188	3.78
" 9	3	3200	1013	15.5680	0.7056	4.53
" 10	4	4000	1011	16.2400	0.5425	3.34
" 11	5	2500	1014	15.8375	0.7131	4.50

TABLE XV.—NITROGEN INGESTED (IN GRAMS).*

Date.	Day of observation.	Ham.	Bread.	Milk.	Red wine.	Total nitrogen.
June 7 & 8	1 & 2	12.2257	6.3503	2.8525	0.1225	21.5510
" 9 & 10	3 & 4	12.2257	6.3503	2.8175	0.1225	21.5160
" 11	5	12.2257	6.3503	2.6162	0.1225	21.3147

* Tea, rum and sugar not analyzed.

TABLE XVI.—NITROGEN BALANCE (IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
June 7	1	49.500	21.5510	14.4812	1.6595	16.1407	+5.4103
" 8	2	49.400	21.5510	16.3590	1.6595	18.0185	+3 5325
" 9	3	49.200	21.5160	15.5680	1.6595	17 2275	+4.2885
" 10	4	50.000	21.5160	16.2400	1.6595	17.8995	+3.6165
" 11	5	49.600	21.3147	15.8375	1.6595	17.4970	+3.8177

Relation of the Neusser's granules in the leucocytes: Altogether the granules were very few in all the different forms. Most of the white corpuscles are entirely free from the granules, and many almost entirely free. During the period that

the patient was under observation there was no apparent variation in the richness of the granules. On the other hand, the alloxuric body nitrogen was both absolutely and relatively high (0.67, 0.71 gram per day).

Observation VII.—A man, 20 years old, with physical signs of a commencing left-sided pulmonary tuberculosis. No tubercle bacilli were found in the sputum, however, after repeated examinations. His weight on May 26th was 57 kilo; he was fairly well nourished. During the period of observation he was free from fever. Leucocytes were 10,000 per cmm.; hæmoglobin 70 per cent (Fleischl).

The daily diet was as follows: Ham, 300 grams,; white bread, 450 grams; milk, 500 grams; wine, 500 grams; tea (infusion), 500 grams; rum, 30 grams; sugar (6 pieces), 30 grams; Rohitsch water, 2 bottles.

The above food represented about 2370 calories (41 per kilo body weight), which were made up as follows:

Albumin	515.65 calories.
Fat	385.02
Carbohydrates	1161.38
Alcohol	308.00
Total	2370.05

TABLE XVII.—URINE.

Date.	Day of observation.	Daily amount in ccm.	Specific gravity.	Total nitrogen in grams.	Alloxuric body—N in grams.	Percentage of alloxuric body—N of the total nitrogen.
May 27	1	4200	1010	15.5820	0.6064	3.89
" 28	2	3450	1012	19.6822	0.5796	2.89
" 29	3	2700	1013	21 3097	0.5953	2.79
" 30	4	3000	1012	18.7950	0.5853	3.11
" 31	5	3400	1014	20.3490	0.5533	2.72
June 1	6	3350	1012	20.2842	0.5803	2.86

TABLE XVIII.—NITROGEN INGESTED (IN GRAMS).*

Date.	Day of observation.	Ham.	Bread.	Milk.	Wine.	Total nitrogen.
May 27 & 28	1 & 2	15.3515	5.8262	3.9094	0.0350	25.1220
" 29	3	15.3515	5.8262	2.7956	0.0350	24.0083
" 30	4	17.4240	5.8262	2.7956	0.0350	26.0808
May 31 & June 1	5	17.4240	5.8262	2.9198	0.0350	26.2050

* Tea, rum and sugar not analyzed.

TABLE XIX.—NITROGEN BALANCE (IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
May 27	1	57.700	25.1222	15.5820	1.2634	16.8454	+8.2763
" 28	2	56.200	25.1222	19.6822	1.2634	20.9456	+4.1766
" 29	3	55 400	24.0083	21.3097	1.2634	22.5731	+1.7352
" 30	4	56.200	26.0808	18.7950	1.2634	20.0584	+5.9214
" 31	5	56.200	26.2050	20.3490	1.2634	21.6124	+4.5826
June 1	6	56.200	26.2050	20.2842	1.2634	21.5476	+4.5574

Relation of Neusser's granules in the leucocytes: The granules are altogether extremely numerous, particularly in the small mononuclears. There was no appreciable variation in the granules from day to day. In this case, with the large number of granules, there was found to be an elimination of a large quantity of alloxuric body nitrogen.

Observation VIII.—The patient was a woman 45 years of age, with cirrhosis of the liver. Since 1895 she had complained of gastric symptoms; pain in the epigastrium. Had emaciated considerably. Had been jaundiced. The gastric juice showed a very marked acidity; free and combined HCl 0.35 to 0.41 per cent, and free HCl alone 0.22 to 0.28 per cent. The liver was enlarged, hard and nodular. Spleen was not much enlarged, but was hard and could be palpated at the costal margin. Body weight averaged about 60 kilos. Vomiting was frequent and patient was moderately anæmic.

The daily diet was as follows: Bread, 300 grams; milk, 500; wine, 500; tea (infusion from 6 grams), 250 grams; rum, 30 grams; sugar (6 pieces), 30 grams; Rohitsch water, ½ bottle.

These food stuffs yielded about 1752 calories (28 calories per kilo body weight), which were made up as follows:

Albumin	283.31 calories.
Fat	233.43
Carbohydrates	927.33
Alcohol	308.00
Total	1752.07

TABLE XX.—URINE.

Date.	Day of observation.	Daily amount in ccm.	Specific gravity.	Total nitrogen in grams.	Alloxuric body—nitrogen in grams.	Percentage of alloxuric body—N of the total nitrogen.
June 24	1	1350	1023	12.4504	0.4831	3.87
" 25	2	1000	1022	10.7800	0.4498	4.17
" 26	3	550	1027	8.1428	0.4591	5.63
" 27	4	850	1025	11.5133	0.5087	4.32
" 28	5	1050	1020	11.0618	0 5375	4.85
" 29	6	1500	1018	11.1938	0.4594	4.10

TABLE XXI.—NITROGEN INGESTED (IN GRAMS).*

Date.	Day of observation.	Ham.	Bread.	Milk.	Wine.	Total nitrogen.
June 24	1	8.8242	5.6056	2.7037	0.0350	17.1685
" 25	2	6.3491	5.6056	2.7037	0.0350	14.6934
" 26&27	3 & 4	4.8425	5.6056	2.6095	0.0350	13.0926
" 28&29	5 & 6	4.8425	5.6056	2.8875	0.0350	13.3706

* The variation in the nitrogen in the ham was due to the fact that the patient could not continue to take the full quantity (220 grams) that she was first given.

TABLE XXII.—NITROGEN BALANCE (IN GRAMS).

Date.	Day of observation.	Body weight.	Nitrogen ingested.	NITROGEN ELIMINATED.		Total nitrogen eliminated.	Balance.
				Urine.	Fæces.		
June 24	1	60.700	17.1685	12.4503	1.3508	13.8011	+3.3674
" 25	2	60.400	14.6934	10.7800	1.3508	12.1308	+2.5626
" 26	3	59.600	13.0926	8.1427	1.3508	9 4935	+3.5991
" 27	4	59.900	13.0926	11.5132	1.3508	12.8640	+0.2286
" 28	5	59.700	13.3706	11.0617	1.3508	12.4125	+0.9580
" 29	6	59.500	13.3706	11.1937	1.3508	12.5445	+0.8261

Relation of Neusser's granules in the leucocytes: The granules were very numerous, especially in the mononuclears,

although there was considerable variation in the granules from day to day. As will be seen from the table there was a large amount of alloxuric body nitrogen eliminated.

These observations that have been made do not in any way confirm the theory that there is a regular coincidence between an abundance of the granules and an increased excretion of the alloxuric bodies, nor do they favor the view that Neusser's granules occur exclusively in patients with an alloxuric diathesis. Cases occur in which, with numerous granules in the leucocytes, there is a relatively small amount of the alloxuric bodies eliminated in the urine, while on the other hand the granules may be almost entirely absent and the alloxuric bodies be excreted in increased amount. Such directly opposed conditions may occur in different individuals having the same disease, as in the two cases of hypertrophic cirrhosis of the liver. Not infrequently the granules become increased or diminished in the same person without there being a corresponding increase or diminution in the excretion of the alloxuric bodies. In fact, increase in the number of granules may be accompanied by a diminution of the alloxuric bodies and *vice versa*.

From the information obtained by the study of the above cases one seems justified in concluding that the supposed relationship between the perinuclear basophilic granules and an alloxuric diathesis, as claimed by Kolisch, is purely empirical.

In conclusion, I must thank Professor Kraus and his assistants for many kindnesses and for valuable aid in carrying on the above analyses.

ENCYSTED DROPSY OF THE PERITONEUM SECONDARY TO UTERO-TUBAL TUBERCULOSIS AND ASSOCIATED WITH TUBERCULAR PLEURISY, GENERALIZED TUBERCULOSIS AND PYOCOCCAL INFECTION.

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The following case is of interest not so much because of lesions in themselves unique, as because of the rare combination of many lesions, the extensive character of these lesions, and the multiple nature of the infection.

The case has many features in common with that reported by Gardner,[1] of Montreal, in the year 1885, and is similar to those described by Wm. T. Howard,[2] of Baltimore, the same year.

CLINICAL HISTORY.

N. A., primipara, aged 30 years; colored. Admitted to the Maternité of the Hospital of the Good Samaritan, in the service of Dr. B. B. Browne, November 19, 1894.

The patient had been married several years; had no children, no miscarriages. Menstruation commenced at the age of 15; it had always been regular, painful and profuse, lasting from six to eight days, during which time she was compelled to remain in bed. Her last period occurred about the middle of March, 1894.

Past History. No family history could be obtained. The patient had measles after reaching adult life. Five years ago she suffered with an attack of "malaria" which lasted three months; at this time she claims to have been "very sick." The attack recurred in September, 1894.

History of Present Condition. The patient is in the eighth month of her pregnancy. Throughout this time she has been feeling ill; she has had pain in the back and abdomen, increased on exertion. At present there are constant backache, and pain in the lower part of the abdomen. Appetite is good; bowels constipated; urine of low specific gravity, otherwise normal.

Examination. Fœtus occupies the left occipito-posterior position. In the right iliac region is a mass, partly soft, partly resistant, which seems to be connected with the uterus, and is not painful except on deep pressure.

On November 18 at 9.30 p. m. the woman was delivered of a healthy child, but owing to uterine inertia the placenta had to be removed. The labor was protracted, lasting 38 hours. The perineum was lacerated and repaired at once. Two days after delivery the patient was attacked with a chill followed by fever and sweating. The record of the puerperium shows an irregular temperature of septic character whose highest point was 104.3°, whose average was 102°. There was no fever after the second week and the patient's condition was recorded as good.

On January 19, 1895, she was discharged from the hospital apparently in fair health. The following April the patient visited the hospital clinic for treatment, considering herself again pregnant since February. In July, 1895, she returned with enlarged abdomen and general discomfort. At this time she claimed to have felt the fœtal movements; in August she was seen at the clinic by Dr. B. B. Browne, who, after examination, did not think her pregnant.

Second Admission. In October, still believing herself pregnant, the woman was readmitted to the Maternité. After careful examination under an anæsthetic, pregnancy was definitely excluded from the diagnosis. At this time a large hard mass was found to the right of the uterus and apparently connected with it. The nature of the tumor could not be determined. Following the ether examination the woman became quite ill. There was great pain in the lower part of the abdomen and sudden rise of temperature. The patient was then transferred to the gynecological department of the Hospital of the Good Samaritan. After the second day the abdomen began gradually to swell, and on December 1st it was much distended. The patient was losing flesh and strength. The temperature showed a typical hectic range, being normal or subnormal in the morning, with an afternoon rise fluctuating between 100° and 103°. The lowest point reached was 95.4°, the highest point 104°.

Repeated examinations of the sputum showed no tubercle bacilli.

Diagnosis. Encysted dropsy of the peritoneum, probably tubercular.

Operation. For removal of the dropsical fluid. On February 26th a laparotomy was performed by Dr. Browne. An incision 5 cm. long was made in the median line of the abdomen. From this opening about eight litres of transparent, pale greenish fluid escaped. The entire anterior portion of the peritoneal cavity was found to be converted into a suppurating cyst. An additional litre of semi-solid caseous material resembling masses of congealed fat was removed by the hand of the operator from the lower part of the sac where it covered the pelvic viscera on the right side. After evacuation of the fluid the surface of the peritoneum was found thickened and converted into a necrotic membrane resembling the caseous masses which floated in the pus.

Examination of Fresh Abdominal Fluid. The macroscopic appearance was that of a turbid, pale greenish yellow fluid in which floated shreds and flakes of caseous material. On standing it separated into two layers, an upper transparent, greenish fluid and a lower dense, creamy mass. The microscopic examination of this fluid showed numerous pus cells, some red blood corpuscles and shreds of necrotic tissue entangling pus cells. Stained cover-slip preparations of the fluid exhibited numerous cocci in pairs, clusters and short chains, but no tubercle bacilli were found.

Immediately following the operation the patient's condition was much depressed. She rallied, however, responding to stimulation. Her general condition improved and she became much more comfortable until the tenth day, when without pain or other distressing symptom she died suddenly, March 6, 1896. The autopsy was made three hours after death.

Anatomical Diagnosis. Tuberculosis of the Fallopian tubes, uterus, ovaries, peritoneum, pleura, and viscera generally. Acute fibrino-purulent and caseous peritonitis; acute serofibrinous and hemorrhagic pleurisy; mixed tubercular, staphylococcus and streptococcus infection; congestion of viscera; general arterio-sclerosis.

The body is much emaciated. In the median line of the abdomen is a gaping incision. It begins 3.7 cm. below the umbilicus and extends 5 cm. downward, exposing a sloughing, puriform cavity from which bubbles of gas are evolved on pressure. The subcutaneous fat is quite absent; the muscles are brownish red in color.

Abdomen. On opening the abdomen the entire anterior portion of the peritoneal cavity is found to be converted into a suppurating cyst. This extends from the liver above to the pelvis below, and traverses the lateral diameter of the abdomen from flank to flank, dipping deeply on both sides. The walls of the sac are made up of dense, opaque, yellow necrotic material about 5 cm. in thickness. The cavity contains a small amount of the same puriform material found at operation. Extending across the cavity obliquely downward, and from before backward, as if to support its somewhat flaccid walls, are four or five dense fibrous bands covered by caseous material, continuous with that lining the general suppurating sac. Of these the largest is in the median line. It is long and almost cylindrical, measuring 3.7x2.5 cm. in diameter. Its anterior and upper attachment is to the abdominal wall 2 cm. below the umbilicus. Its posterior attachment below is by a broad expansion upon the posterior wall of the sac where it dips down to cover the pelvic viscera. A second organized band, smaller, but similar in character to the one just described, is found on the left side. A few adhesions are also found in the posterior wall of the sac on the right side. On transverse section these bands show an organized, pink, fibrous groundwork thickly inlaid with miliary tubercles and limited by a zone of caseous material. The posterior wall of the pus sac is found so densely adherent to the intestines and other adjacent viscera that separation is difficult. No trace of the normal omentum can be found, and the probabilities are that it enters largely into the composition of the suppurating cyst and the traversing fibro-caseous bands.

The coils of intestine are densely matted together by old fibrous bands and by more recent and lighter adhesions, studded everywhere with yellow miliary tubercles, and form a single compact mass filling the greater part of the abdominal cavity. Not only are the intestinal coils adherent to each other, but they are bound to all neighboring viscera. The mesentery is greatly thickened and contracted, and is thickly infiltrated with yellow miliary tubercles.

The mesenteric lymph glands are enlarged, indurated and caseous.

Upon separation of the more delicate adhesions between the upper coils of intestine, several smaller cysts about the size of a hen's egg are found. They contain clear straw-colored fluid. There is a slight excess of clear fluid in the posterior peritoneal cavity, which appears to have been more recently infected.

Liver. The liver measures 35x17.5x8.7 cm. in its various dimensions. The capsule is much thickened, and strong adhesions unite it firmly to the diaphragm. The left lobe is drawn out into a tongue-like process, which extends completely across the abdomen, covering the spleen on its anterior and left lateral surfaces. So completely adherent are these two organs that only on section can a line of organized union be made out. The lower surface of the liver is also in contact with the upper wall of the pus sac, with the stomach and with the intestines, to which it is bound by firm adhesions. On breaking up these adhesions and freeing the surface of its caseous membrane, the capsule is found thickly beset with yellow miliary tubercles. The surface is mottled. On section the parenchyma shows the characteristic appearance of nutmeg liver. Scattered throughout its substance are numerous tubercles, both grey and caseous. They are mostly submiliary, but larger ones exist.

Spleen. The spleen is considerably enlarged. It is densely bound down by old fibrous adhesions to all neighboring structures. Its anterior and lateral surfaces are almost completely concealed from view by the tongue-like expansion of liver. The capsule is irregularly thickened and contains caseous miliary tubercles. The consistence is much diminished. It tears readily. On section it is congested and contains grey and yellow tubercles, miliary, submiliary and conglomerate in form.

Kidneys. The kidneys are slightly enlarged; the capsules are somewhat thickened, but strip off with moderate ease. The surface of the right kidney shows a shallow circular scar-like depression about 12 mm. in diameter, somewhat paler than the surrounding cortex. Under the capsule of this kidney is also seen a solitary caseous tubercle about the size of a split pea. Upon section the kidneys are congested, somewhat increased in consistence, and contain an occasional isolated tubercle of large size. The circular depression seen upon the surface of the right kidney is found to be the base of a dense, pale, pyramidal area which extends quite down through both cortical and boundary zones, and contains in its centre a small cyst.

Ureters. The ureters are normal, except for a slight dilatation of the right ureter in its upper portion.

Pancreas. The pancreas is pink and firm. Its capsule is thickly studded with miliary and conglomerate tubercles; none, however, can be seen in its substance.

Stomach. The stomach is adherent to all adjacent structures. Its mucous membrane shows congestion, most marked along the rugæ. Some ecchymoses are found at the cardiac end.

Intestines. In some places the felt-like adherent sac wall forms a partial covering to the intestines. Beneath it, as elsewhere, the peritoneal coat is tolerably smooth, congested, and contains numerous miliary and conglomerate tubercles. The mucous membrane is more or less congested throughout the entire extent. The congestion is most intense upon the valvulæ conniventes. There are no tubercular ulcers, but in places tubercles can be seen extending inward from the peritoneal coat. The walls of the large intestine are thin and very deeply congested. The abdominal lymph glands are enlarged and caseous.

Thorax. The sternum and costal cartilages cannot be readily lifted because of adhesions between the two layers of the pleura on the right side.

Left Pleural Cavity. The left pleural cavity contains about 2.5 litres of blood-stained fluid with flocculi of fibrin. There is one dense organized band of tissue connecting the two layers of the pleura (about the region of the seventh rib). There are also a few delicate adhesions in the posterior and lower part of the cavity. The apex is firmly adherent to the chest wall. The lung is compressed by the excess of fluid in the pleural cavity. Both layers of the pleura present a coarse reticulated mottling, as though a loose-meshed network of fibrin had been laid upon a dark red hemorrhagic background. Tubercles are also seen.

Upon the parietal pleura, as it covers the sixth rib, are seen two softened caseous tubercles, each about the size of a split pea. Cover-slip preparations made from one of these immediately at autopsy show the presence of tubercle bacilli and micrococci in large numbers.

Left Lung. The left lung is compressed and atelectatic, except the anterior margin of the upper lobe, which contains a little air. Scattered irregularly and sparsely through the lung substance are miliary and conglomerate grey and caseous tubercles. No cavities nor old tubercular foci can be made out.

Right Pleural Cavity. The right pleural cavity is dry. The lung is bound down throughout its entire extent by easily detached adhesions. At the base and posteriorly the adhesions are pretty firm.

The pleura is covered with a layer of fibrin of irregular thickness, more or less organized at the base of the lung. It contains caseous tubercles.

Right Lung. The right lung is also considerably contracted by the upward pressure from the abdomen and the lateral pressure from the left thorax. The lower lobes are atelectatic and congested. The upper lobe contains a little air and exhibits along its anterior margin a few emphysematous patches. The distribution of tubercles in its substance corresponds with that of the left lung.

The bronchial and mediastinal lymph glands are enlarged and caseous.

Heart. Owing to the excess of fluid in the left pleural cavity, the heart occupies a position behind the sternum, almost in the median line. The pericardium, the pericardial fluid, the endocardium and heart valves are normal. The aorta contains thickened yellow atheromatous plaques.

Cranial Cavity. There is a slight increase of cerebral fluid. The meninges of both cerebrum and cerebellum are congested, particularly on the under surface. The pia arachnoid at the base of the cerebellum contains two or three yellowish white bodies resembling miliary tubercles.

Cover-slip preparations made from these nodules show no tubercle bacilli.

The pelvic viscera were removed at autopsy and placed in formalin, for detailed description later.

Pelvic Viscera. Uterus. The uterus is 8.5 cm. long, 5 cm. broad and 2.5 cm. in the antero-posterior diameter. Both the anterior and posterior surfaces are covered by an opaque, yellowish white, felt-like membrane, varying from 2 to 6 mm. in thickness. This membrane passes directly from the uterine walls to the other pelvic viscera, forming a complete blanket, and constituting the lower part of the large pus sac which occupied the anterior portion of the peritoneal cavity. Projecting from the middle third of the posterior wall is a myomatous nodule about the size of a large walnut, attached to the uterus by a short and broad pedicle, Fig. 1, M. Springing from the fundus of the uterus about 2 cm. anterior to the right cornu is another pedunculated myoma, large, ovoid, and 9.5x7.5x5.5 cm. in diameter, Fig. 1, M. From the left surface of this tumor springs a cylindrical band about 3 cm. long and 13 mm. in diameter, attaching it loosely to the coiled-up mass of intestines behind. This band is of the same general nature as the fibro-caseous bands which traverse the abdominal cyst. Another band, broad, flattened, and 6 mm. thick, is attached to the right surface of the tumor. This forms part of the general peritoneal sac. On stripping back the dense, felt-like membrane covering these tumors, their surfaces are found studded with tubercular nodules of a yellowish white color, and from one to three mm. in diameter. The smaller tumor can be easily shelled out of its capsule, owing to a peculiar orange-yellow caseous material separating the two in the region of the pedicle. The myomata, so far as can be seen, do not contain tubercles in their interior. The uterine wall measures 1 cm. in thickness at the fundus, Fig. 1, F. U., 2 cm.

anteriorly and 1 cm. posteriorly. It is pale pink in color and is studded with sparsely scattered, yellowish, miliary and conglomerate nodules.

The cervix is 2.5 cm. in length. The mucous membrane of the body presents a shaggy, moth-eaten appearance, of yellowish color and friable consistence. The mucous membrane of the cervix, which is much less rough and friable, still presents indications of the normal rugæ. The mucosa varies from 2 to 5 mm. in thickness.

Right Appendages. Springing from the right uterine cornu and extending backward and downward, so as to be completely concealed from view by the large myomatous tumor which filled the right inguinal region, is a mass 8 cm.x5.5 cm.x2.5 cm. in size. It presents at first sight the appearance of an enlarged ovary, covered by the general pyogenic membrane, beneath which, as in the uterus and elsewhere, are miliary and conglomerate tubercles studding the surface. Upon section, however, it is seen to consist of the convoluted tube, thickened and necrosed, with its exaggerated coils held together in a stroma of dense organized connective tissue studded with miliary tubercles. Beneath the lower part of this convoluted mass is seen a portion of the ovary, so completely covered by and adherent to the tube as almost to have lost its own identity, Fig. 1, r. Ov. A longitudinal section through this tubo-ovarian tumor gives transverse, oblique and longitudinal sections of the much distorted tube. They lie like scattered caseous islands with ulcerated centres, in the tubercle-dotted stroma. The walls of the tube are from 3 to 8 mm. thick, and are for the most part uniformly caseous. Here and there is a nodular studding which gives the mucosa an uneven surface; or again a rich infiltration of the other coats with barely agglomerated caseous tubercles is observed. The fimbriated extremity, dilated, and curled outward in trumpet-like expansion, is directly continuous throughout its circumference with the anterior peritoneal sac, Fig. 1, r. F. E. The folds of the fimbriated end are everywhere visible and appear densely thickened and ragged; the extremity itself measures 2.5 cm. in diameter.

Left Appendages. The left tube is exceedingly tortuous, and from the uterine cornu outward gradually increases in size, terminating in a fimbriated extremity 4.5 cm. in diameter. It is 11 cm. long in its contracted state, Fig. 1, F. E. The folds of the fimbriæ are transformed into a greatly thickened, moth-eaten membrane, from whose surface project numerous papillary processes varying in size and shape and measuring from 10 to 14 mm. in length. The interior of the tube bears some general resemblance to the necrotic membrane which covers its surface and which represents the walls of the abdominal sac with which both tubes appear to be directly continuous. The convolutions of this tube, while excessive, as are those of the right side, are not embedded in a dense organized stroma, but are held together by tolerably firm adhesions. These, as well as the surface of the tube, exhibit caseous, miliary tubercles, Fig. 1, l. T. Longitudinal and transverse sections through the tube show extreme necrosis of its walls. They are more extensively ulcerated toward the outer third, where the condition is most advanced. The lumen at this point is increased to 13 mm. in diameter. The walls of the tube are here from 3 to 5 mm. thick and consist mainly of caseous material with a narrow outer border of organized tissue. This forms a capsule which can be readily stripped away from the central necrotic mass. The thickness of the tube about its middle portion is 2 cm. There is no lumen visible on cross section, but the entire tube seems made up of one dense, yellowish, homogeneous mass.

The left ovary lies behind and beneath the tube, to which it is bound by adhesions similar to those connecting the convolutions of the tube, Fig. 1, l. Ov. From this it may be detached with moderate ease. It is about normal in size and position. The surface is covered by the same necrotic membrane, and exhibits upon its removal sago-like bodies. On section the structure is smooth, firm, grey and glistening, showing several corpora fibrosa, and one flattened tubercle just beneath the surface.

Connected with the pelvic viscera and binding them together are portions of the peritoneum which went to make up the original sac wall but which were torn away at autopsy. They are of the same general appearance as the necrotic membrane covering the viscera. A similar sac, collapsed and dipping down behind and to the left of the cervix uteri, is covered in by the small myoma above described. This represents the remains of an abscess about the size of an orange which ruptured at autopsy, discharging thick, greenish yellow pus.

The Vagina. There are several small, greyish, flattened elevations upon the surface of the mucous membrane, irregular in size and shape, some round, measuring from 1 to 2 mm. in diameter; others with irregular edges measuring about 6x3 mm. in diameter. There is also a small superficial ulcer in the posterior vaginal wall just below the cervix. It is about the size of a split pea, has sharp irregular edges, but contains no tubercles in its walls. The vagina is otherwise normal.

The *bladder* is contracted and is apparently normal.

The *rectum* is partly covered by necrotic membrane, but shows no abnormalities.

Histological Examination.

Pelvic Viscera.

Uterus. The surface epithelium has entirely disappeared, and the uterine mucosa shows a granular necrosis throughout the inner fourth of its thickness. Beneath this the normal stroma is invaded by diffuse tubercle tissue with discrete caseating nodules. It is the union of these nodules near the surface of the mucosa which results in the granular necrosis. In the depth of the membrane the uterine glands are preserved, although the epithelium is swollen and invaded by mononuclear and polymorphonuclear cells. They are often dilated, and contain swollen desquamated epithelial cells, polymorphonuclear leucocytes and granular detritus. The diffuse tubercle tissue consists largely of lymphoid and epithelioid cells, with an occasional giant cell. The discrete caseating nodules present the typical structure of caseous tubercles. The giant cells of both stroma and nodule are round, oval or irregular in shape, with a mural or polar arrangement of the nuclei. The epithelioid cells of the diffuse tubercle tissue are usually round or polyhedral, with small vesicular nuclei. Those of

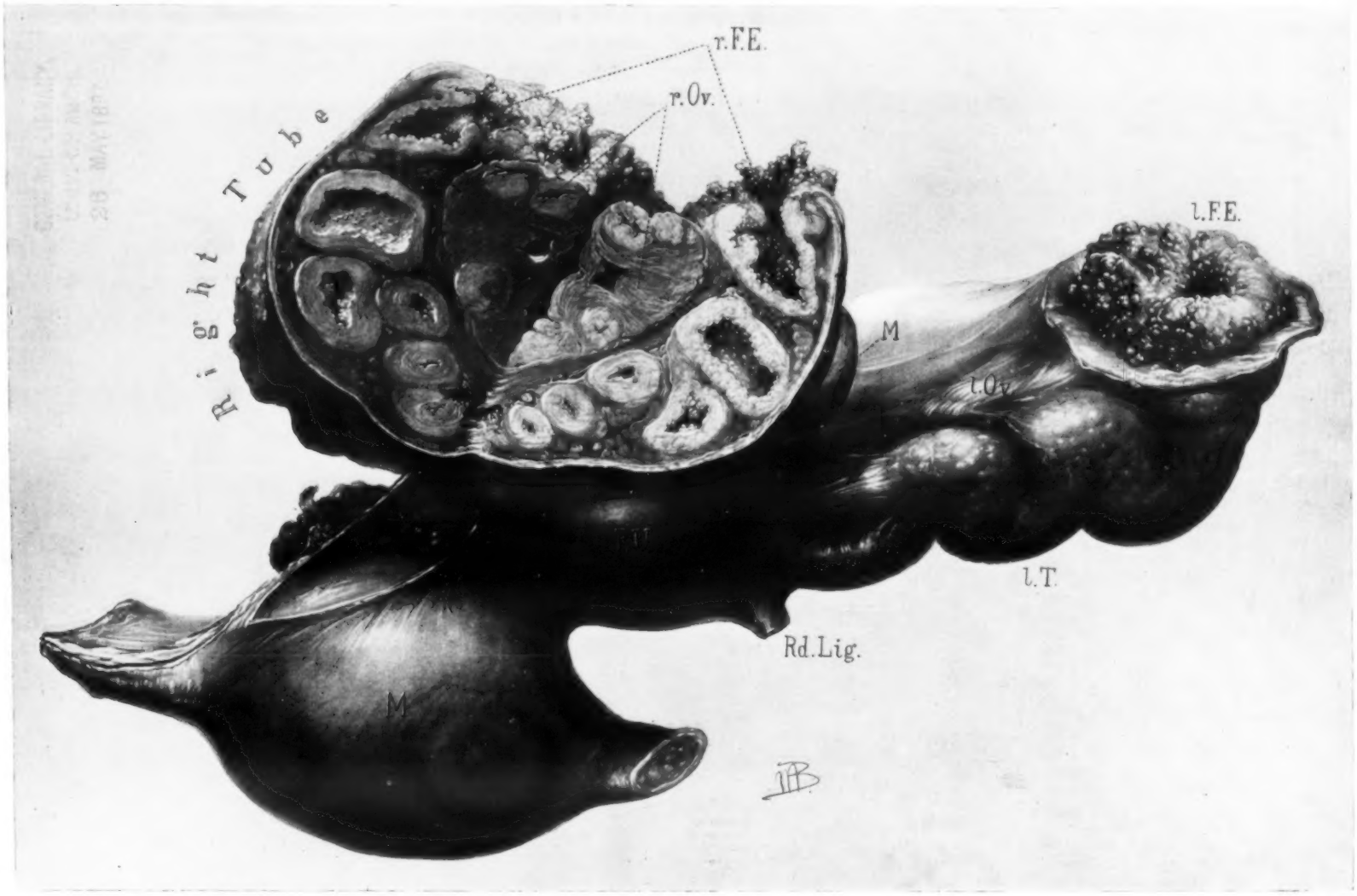

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FIG. 1.

the caseating nodules are irregular and elongated, with oval, bizarre and wavy nuclei, the close packing of which in the periphery of the nodule gives the appearance of a radiating fringe.

The muscular coat is infiltrated throughout its entire thickness with diffuse tubercle tissue and discrete miliary nodules, most of which are caseous. The diffuse infiltration is represented by small cells with solid and vesicular nuclei, arranged in longitudinal strands between the muscle bundles. The caseous nodules lie next to the peritoneal surface, and are irregularly triangular in shape, with bases toward the serosa. Sometimes the bases coalesce and form a continuous scalloped caseous zone. The muscle cells are increased in number, and here and there are myomatous foci which consist of dense aggregations of cells containing elongated club-shaped nuclei.

The blood-vessels of the muscular coat present interesting features; some of them show the typical changes of endarteritis obliterans, with more or less encroachment upon the lumen. But the most characteristic lesion is the tubercular involvement of the vessel walls. This process sometimes begins in the intima and remains limited to this coat, or extends to the media or adventitia. Sometimes it takes origin in the adventitia and perivascular tissues, and spreads inward in like manner through the other coats. It may be diffuse or circumscribed, it may be a cellular proliferation simply, or show all stages of degeneration, from an early nuclear fragmentation and karyolysis, to advanced caseation in which all the coats and even the vessel contents are transformed into a dense hyaline mass. The corpuscular contents of the vessels are often preserved intact, notwithstanding advanced changes in the vessel walls. Again there are thrombi, hyaline or mixed, in various stages of transformation. Sometimes the vessels are the centre of a tubercular process, in which case their fibrinous contents are continuous with a fibrillated fibrin which extends out into the surrounding tissues. Where the tubercular change is early and begins in the intima, the endothelium is sometimes preserved intact; here there is subendothelial proliferation of the connective tissue cells, forming epithelioid cells of a round or polyhedral shape, with moderate or abundant protoplasm and small round nuclei. But in other cases there is evidence of endothelial proliferation, these cells enlarging, becoming at times cuboidal, and forming a lining of two or more rows to the lumen, with occasional complete occlusion.

In the serous membrane the changes are most marked; on the surface is a dense, opaque, necrotic coat, granular, fibrillated and hyaline. Beneath this is a narrow zone of nuclear fragmentation, while yet below and forming a line of demarcation between the necrotic portion of the serous coat and the scalloped caseous zone of the muscular coat is a narrow band of œdematous, vascular connective tissue containing a few muscle fasciculi, and showing infiltration with lymphoid, epithelioid, polymorphonuclear and occasional giant cells.

The process is least advanced in the cervix, most advanced in the fundus. In the cervix there is no invasion of the muscular coat, and the vaginal portion contains no evidence of tubercular involvement other than a solitary cellular tubercle under the stratified epithelium in its upper part, and a cellular proliferation of the corium, gradually disappearing as the outlet is approached.

Among the diffuse tubercular tissue are found numerous homogeneous, highly refractive globules of various sizes, occurring singly or in groups. They show especial affinity for acid aniline dyes, and also stain intensely with gentian violet. In shape they are invariably round; in size they vary from that of a micrococcus or a basophilic granule to that of a lymphoid cell, some even exceeding this. Their average diameter is that of a red blood corpuscle; in fact, when uniform in size, and occurring in masses, they are readily mistaken for such in specimens which have been stained with hæmatoxylin and eosin. The little globules are, however, more homogeneous and solid and more highly refractive than red blood corpuscles. These appear oftenest more or less closely packed within cell bodies, the nuclei of which are still preserved. Sometimes they accompany a more advanced degeneration, in which case the nucleus stains poorly or not at all. Again, they are free from cellular inclusion, and lie in groups or scattered through the tissues. The groups are circular, elongated or irregular, and contain from two to thirty or more members. With Weigert's fibrin stain they stand out conspicuously as deep blue spheres, homogeneous and structureless. With Russell's fuchsin stain they strike a bright red hue; with eosin they stain pink. In many respects they correspond with the hyaline bodies considered by Lubarsch[9] as a form of albuminous degeneration, found by various observers in both normal and pathological conditions, and described by Russell[10] as "Fuchsin bodies." In the present case they contain neither nuclei nor spores such as Russell and others have described.

In all three coats of the uterus micrococci are found arranged in pairs, clusters, or chains of greater or less length, numbering from three to sixteen members. They are especially numerous along the superficial caseous zone of the peritoneal coat, where they form a thick, irregular border and extend deep into all fissures of its necrotic structure. In the muscular coat they are less numerous, and are found for the most part within and about blood-vessels in the bands of the infiltrated tubercle. In the mucous membrane they are again present in considerable number, somewhat sparsely scattered at times or occurring on the surface as dense aggregations. Where scattered, their arrangement is usually in the form of pairs and chains.

Tubercle bacilli are also found in the uterine tissues. They occur in the interior of vessels, either free or enclosed within cells, in the vessel walls, and in the tubercle tissue outside.

The uterine myoma is covered by newly formed connective tissue, with rich infiltration of diffuse and circumscribed tubercles and many pus cells. The capsule is made up of strata of well preserved fibro-muscular bands. But the interior of the tumor is necrotic throughout. Near the surface of the necrotic myoma is a narrow zone of calcareous matter; the walls of all blood-vessels in its vicinity are likewise calcified. A still broader zone of necrotic myomatous structure, including the calcareous band, is dotted over with spiculated spherules of golden yellow pigment. No tubercles are seen. Russell's fuchsin bodies are numerous in the tissue covering the myoma, but none are found in its substance. Micro-organisms similar

to those found in the uterus are likewise found in the tubercular covering of the tumor.

Right Fallopian Tube. The mucous membrane of the right Fallopian tube at its uterine end has an almost intact epithelium. In one place, however, the epithelium is wanting, and here the stroma is invaded by a mass of tubercle tissue, some of which has broken off and lies free and degenerating in the centre of the lumen, together with the desquamated epithelium. The middle coat of this part of the tube retains its muscular structure; bands of muscle, however, are separated from each other by serous infiltration or by alternating bands of diffuse tubercle tissue. The serous coat is composed of solitary caseous tubercles so blended as to form a continuous scalloped zone. On passing outward from the uterine end the tubercular process becomes more marked, and when the middle portion of the tube is reached the walls are so intensely involved as to form a thickened necrotic mass, with no evidences of structure except a narrow fibro-cellular zone encircling the tube about 2 mm. from the periphery. The necrotic substance is hyaline in the centre, where it is pierced by a ragged slit-like lumen. It corresponds here with the remains of the mucous membrane. There is nuclear fragmentation within the fibro-cellular zone, and among the nuclear fragments isolated bands of muscle are preserved in scanty number. The surface necrosis corresponds essentially with that of the uterine serosa, while the middle fibro-cellular zone is continuous with the thickened, œdematous, vascular connective tissue of the uterus, which there formed a line of demarcation between the necrotic portion of the serous coat and the scalloped caseous zone of the muscular coat. Here, as there, it contains a few isolated muscle bundles, the only normal elements of the original Fallopian tubes. Here, as there, it is infiltrated with tubercular cellular tissue and polymorphonuclear cells, and here, as there, it shows many vascular changes, both sclerotic and tubercular, similar to those described in the uterine muscular coat.

There are numerous dilated lymph spaces and blood-vessels with subendothelial proliferation or a lining of cuboidal cells. Occasionally a well-defined giant cell takes origin in the endothelium of a vessel and projects into the lumen, filling it more or less completely. Sometimes such a cell is found free in the vessel. Indeed, the giant cells are numerous throughout the middle coat, and are especially abundant near the necrotic mucosa. They are often situated in little caverns, which are sometimes surrounded by lymphoid cells. Where not so situated, they are more apt to be surrounded by an irregular zone of epithelioid cells. They are elongated or irregularly ovoid in shape, of various sizes, and contain nuclei at their poles or about the periphery, seldom in the centre. Sometimes they are invaded by polymorphonuclear and lymphoid cells.

In the fimbriated extremity the degeneration is extreme. The lumen is considerably dilated, the walls are convoluted and thick. The mucous membrane is composed of three strata representing gradations of tubercular change. Next to the lumen is a narrow necrotic border, which takes an intense eosin stain. It gives occasional evidence of original structure in the preserved outlines of old blood-vessels, of connective tissue bands, of giant cells, and even of an occasional caseous tubercle which has not yet lost its identity by coalescence with the general necrotic border. Outside of this is nuclear fragmentation with structural outlines still better preserved; while yet beyond is the normal stroma invaded by infiltrated tubercle. The middle coat is a comparatively narrow band of fibro-cellular tissue, not yet degenerated, forming less than a third of the whole tubal thickness, and presenting the same interesting features as the corresponding coat of the middle portion of the tube. The thickest part of the wall is the peritoneal coat; it is entirely involved in an extensive necrosis which is at one time fibrillated, at another granular or hyaline.

The little refractive globules described in the cellular tubercular tissue of the uterus as Russell's fuchsin bodies are present also in the tube in considerable number. They lie by preference in the middle coat, in which the tubercle tissue is best preserved. They present the same general characteristics as the hyaline bodies of the uterus, but are somewhat smaller. Indeed, whereas in the uterus their average diameter is that of a red blood corpuscle, in the tube their average size is that of a micrococcus; and, where Weigert's fibrin stain is used, they could readily be mistaken for such, were it not that upon careful examination they show less uniformity in size, take a more solid stain, and because larger globules, both free and in cells, are found in the same neighborhood. Extra-cellular spheres of large size grouped with smaller hyaline globules are sometimes found occupying open spaces in the tissues.

The borders of the necrotic zones, both mucous and serous, in all parts of the tube, are invaded to some depth by deeply staining micrococci, which in form, size, grouping and arrangement in the tissues, resemble the organisms found in the uterine coats. Similar organisms are found at times in the blood-vessels of the middle coat. The streptococci predominate, and the average length of the chains is six members. Tubercular bacilli are likewise found.

Right Ovary. The right ovary is somewhat enlarged. Its surface presents a smooth, wavy outline and is covered by a thick, œdematous, vascular connective tissue which contains lymphoid and epithelioid cells, but no giant cells. There are also numerous dilated lymphatic vessels filled with lymphocytes. The edges of this connective tissue covering are ragged and represent the remains of old fibrous adhesions between the ovary and neighboring structures. Deep within the ovarian stroma are several isolated and conglomerate caseous tubercles. There are many advanced corpora fibrosa, and an unruptured Graafian follicle in the process of retrograde metamorphosis. Russell's fuchsin bodies are numerous in the tubercular tissue covering the ovary, and in the ovarian tissue itself. They are present where the degeneration is least advanced, and may be seen, not only in the tubercular areas, but in association with other degenerative changes—in the hyaline degeneration of corpora fibrosa and in the retrograde metamorphosis of unruptured Graafian follicles.

Streptococci, staphylococci and tubercle bacilli are present. Of these the streptococci are most abundant and occur in long chains, at times reaching the length of fourteen members. They are located within the lumen of blood-vessels, in the blood-vessel walls, or in the perivascular lymphatics of the

connective tissue covering the ovary, and are oftenest free, but may be found enclosed in cells.

Left Fallopian Tube. The left Fallopian tube has the same general structure as the right, the only difference being that the tubercular lesion is more advanced. The middle portion of the tube is converted into a dense necrotic cord, the transverse section of which shows a ragged longitudinal rift in the centre of an oval area of hyaline necrotic material. This is surrounded by a fibro-cellular border, whose surface is roughened by fibrous shreds torn in the process of separation from adjacent structures. The fibro-cellular border corresponds with both middle and outer zones of the other tube. The fimbriated extremity, like its fellow, is extensively degenerated, and the lumen is also much enlarged. Its walls, however, are less uneven and are but half the thickness of those of the corresponding tube. Continuous with the middle portion of the tube the fimbriated extremity likewise has an outer border of fibro-cellular tissue and an inner hyaline necrotic coat. In this tube there are fewer giant cells than in the other, and the tubercle tissue in places shows fibroid change. Russell's fuchsin bodies are present in small number.

The micro-organisms found are similar to those in the other tube.

Thus, while both tubes show in the main the same tubercular lesions, they differ in that the left tube has somewhat thinner walls, contains a smaller number of giant cells, exhibits in places fibroid change, and the thickened necrotic serous coat is wanting.

Left Ovary. The left ovary is smaller than the right and is surrounded by diffuse tubercular tissue. In gland-like spaces near the surface are found great accumulations of nuclei, bound together by hyaline and fibrillated fibrin, and often presenting the appearance of the tubercle giant cell. In this ovary there is but one solitary caseous tubercle, about the size of a split pea, which projects slightly above the surface and dips down into the stroma. Fuchsin bodies and micro-organisms are present, resembling in every respect those found in the right ovary.

The Vagina. The vagina shows no tubercular involvement. The elevated greyish white patches observed on macroscopic examination consist of swollen, distorted, stratified epithelial cells. Occasional polymorphonuclear and mononuclear cells have wandered up into the epithelium. There is slight proliferation of the cellular elements in the upper portion of the corium. Fuchsin bodies are found in these proliferating areas. More densely stained globules are seen in the swollen epithelium above. Upon the surface of the mucous membrane there are a few cocci and long bacilli. Within the blood-vessels of the vaginal wall, and especially in the larger ones, cocci occur in pairs and chains, both within cells and outside of them.

The *bladder* and *urethra* show nothing abnormal.

Pus Sac. The wall of the pus sac, portions of which remain adherent to the pelvic viscera, is from 4 to 6 mm. in thickness. It consists of three coats not sharply differentiated one from the other. In the inner necrotic coat, which represents about one-third of the thickness of the sac wall, the degeneration is extreme. Its surface is edged by dense aggregations of micrococci which in places extend deep into the necrotic structure. The middle coat is fibro-cellular and contains a few isolated clusters of fat cells. It is infiltrated with diffuse tubercular tissue. The outermost coat is not sharply separated from the middle, and differs from this in containing numerous closely packed caseous tubercles. Fuchsin bodies are present as intracellular and extracellular globules. They are of all sizes, and contrary to what occurs elsewhere, they are found in the most advanced necrosis. In such places they take a less intense gentian-violet stain and merge gradually into the surrounding necrotic structure. Micrococci are found in all three coats arranged in pairs, clusters and chains. They are especially numerous upon the inner surface of the sac, where they form a continuous irregular layer. Tubercle bacilli are also present in small number.

General Viscera.

The *lung* shows very little that is abnormal. The visceral *pleura* is considerably thickened and presents an irregular wavy outline. Its surface is covered by a layer of fibrin formed by the coalescence of highly refractive clumps. These, in places, extend deep into the tissues, where they appear as densely hyaline whorls, or the fibrin remains limited to the surface, where it forms a meshwork enclosing polymorphonuclear cells. The more hyaline portions are at times invaded by organizing connective tissue. Beneath the fibrinous covering is a layer of highly vascular granulation tissue which contains closely-set flattened tubercles. These tubercles project here and there between the clumps of hyaline fibrin, extending at times to the surface, or dip down into the lung substance. They are more or less caseous and show the same general structure as the tubercles already described. Where less advanced they contain many giant cells. The deeper layers of the pleura contain dark brown and black granular pigment, both free and enclosed in cells.

The parietal pleura resembles the visceral in its general histological features. It is composed of a superficial layer of fibrin, beneath which are granulation tissue and caseous tubercles. Quite beneath is a third layer consisting of œdematous connective tissue, the meshes of which are widely separated by serum and the spaces of which are occupied by swollen mononuclear cells. In the parietal pleura there are small extravasations of blood. Both layers of the pleura contain the fuchsin bodies and basophilic cells. A few pairs and chains of cocci and tubercle bacilli are present.

Liver. The capsule is thickened by tubercular growth. The lobules show dilatation of the central veins and adjacent capillaries. The hepatic cells in the centre of the lobule contain a brownish yellow pigment. The peripheral cells are filled with fatty globules.

Distributed thickly through the liver substance, in close relation with the blood-vessels, are countless tubercles, most of which are of microscopic size. Large nodules evident to the naked eye are less abundant. The former consist for the most part of lymphoid cells or of lymphoid with a central accumulation of epithelioid cells. The larger tubercles are evidently older and exhibit the typical structure of miliary tubercles with advanced central necrosis. There are a few pairs and chains of cocci in the liver capillaries.

Spleen. The capsule is thickened by a new growth of cellular connective tissue containing caseous miliary tubercles. The spleen parenchyma shows an increase in its cellular elements. Much blood pigment and many red blood corpuscles are found. The arterial walls are considerably thickened, and an occasional hyaline thrombus in one of the smaller vessels is seen. There are numerous tubercles, both young and old, scattered through the splenic substance. Fuchsin bodies are present. Micrococci in pairs, masses and chains are found. They are sometimes arranged within cells, but are usually extra-cellular.

Kidney. The kidney shows parenchymatous and fatty degeneration of the epithelium lining the Malpighian tufts and the convoluted tubules. There is a slight increase of connective tissue, especially under the capsule. The tubules are here compressed, but elsewhere they are at times dilated. Hyaline and granular casts are found. The wedge-shaped area described among the macroscopic lesions is found to consist almost entirely of connective tissue in various stages of development. It compresses the tubules, many of which are completely obliterated; while others persist with intact epithelium or with lumen distended by hyaline casts. The Malpighian tufts are small and fibrous, and in places are converted into hyaline balls. Indeed, the connective tissue invades all structures in the triangular area, and tubules, blood-vessels and glomeruli are alike involved. The central cyst is lined by cuboidal or flattened epithelium. Its walls appear to contain a large number of muscle fibres which have an irregular arrangement and include masses of lymphoid cells. It is continuous with several smaller tubular dilatations and is undoubtedly a retention cyst. At the apex of the triangle is a large dilated blood-vessel with irregular thickened walls and lumen containing remnants of a probable thrombus. The area resembles an infarct in the process of cicatrization, but for the presence of apparently functioning structures. A few cocci arranged in pairs are found within a blood-vessel of the kidney.

Pancreas. The capsule is considerably thickened by a new growth of connective tissue, alternately fibrous, cellular, vascular and œdematous. Numerous closely-set tubercles are embedded in the capsule. They are round and project beyond the surface, or they may be flattened and elongated. They show the typical structure of cellular, caseous and fibroid tubercles. The capsule contains the fuchsin bodies and masses of micrococci.

Lymph Glands. The bronchial lymph glands are invaded by lymphoid, epithelioid and giant cells and show a tendency to fibroid change. Some of the central arteries are sclerotic, others show tubercular lesions. Masses of basophiles are present in the centre of the glands, especially grouped around blood-vessels, and the fuchsin bodies are found at times occupying the interior of basophilic cells. Micrococci occur in pairs and chains within blood-vessels and in the perivascular connective tissue.

In the mesenteric lymph glands the tubercular process is more advanced. The centre is necrotic, with original structural outlines still preserved. The periphery of the gland retains its lymphoid nodules, but these are invaded by tubercle tissue. The blood-vessels are thickened, and basophiles are found. The gland is covered by œdematous tubercular tissue. Diplococci and staphylococci are numerous in the surrounding connective tissue, and beaded tubercle bacilli are found in the interior of the gland.

The *aortic valve*, the *aorta* and the *coronary arteries* show irregular thickening of the intima due to increase of connective tissue which in places is swollen, and the interstices between the branching connective tissue cells are occupied by translucent, highly refractive masses. Or again, the tissue is non-cellular and diffusely hyaline. On the edge of the aortic valve where it appears necrotic a few diplococci are found.

Fibro-caseous bands. The fibro-caseous bands extending across the abdominal pus cavity, and those in the pleural sac, are made up of fibrillated connective tissue, everywhere invaded by diffuse tubercle tissue and circumscribed tubercles in all stages of growth or of retrograde metamorphosis. The peripheries of the bands consist of necrotic zones edged by a closely packed layer of micrococci. The connective tissue stroma shows every stage of organization from young granulation tissue to dense hyaline bands; the blood-vessel walls are thin and easily ruptured, and extravasations of blood are sometimes found. The diffuse tubercle tissue consists for the most part of lymphoid and epithelioid cells grouped usually in the neighborhood of blood-vessels. Few giant cells and some basophiles are found. The tubercles exhibit every stage of growth and of retrograde metamorphosis. At times they present a typical cellular structure with giant cells in the centre. Again, the centre is occupied by a blood-vessel, and the lumen of such vessel may contain well preserved fluid contents, fibrillated fibrin, or a hyaline or mixed thrombus. The caseous peripheral zone is from 2 to 6 mm. in thickness. It contains no cells nor nuclear fragments, but is made up of a structureless necrotic substance, alternately granular, fibrillated and hyaline. Fuchsin bodies are easily demonstrable. They are of all sizes, from that of a basophilic granule to a lymphoid cell, and are both intracellular and extracellular. They occur in the diffuse tubercle tissue where degeneration has just begun. The micrococci found on the surface of the band sometimes extend deep into its substance; they occur in pairs, in masses and in chains. In the bands of the abdominal pus cavity compressed lobules of adipose tissue exist.

Bacteriological Examination.

Cover-slip preparations made at autopsy from a tubercle upon the pleura demonstrated large numbers of long beaded tubercle bacilli and clusters of micrococci. Cultures made from this tubercle enable one to isolate white, lemon yellow and orange colored colonies. Inoculations from these colonies into the various culture media exhibit the peculiar cultural characteristics of the staphylococcus pyogenes aureus, citreus and albus, and cover-slip preparations from the various cultures invariably show cocci arranged in clusters and in pairs. Unfortunately, no cultures could be made from the abdominal viscera, but hardened sections of the organs stained by Gram's, Weigert's and Gabbet's methods exhibit staphylococci, streptococci and tubercle bacilli in all.

SUMMARY OF HISTOLOGICAL AND BACTERIOLOGICAL EXAMINATION.

(1) The condition is a mixed infection due to the tubercle bacillus, the streptococcus pyogenes, and the staphylococcus pyogenes aureus, citreus and albus.

(2) Miliary tubercles, both grey and caseous, are found in all the viscera of the body except the heart and pancreas; in the lymph glands, and in all serous membranes except the pericardium.

(3) Diffuse tubercle tissue is found only in the more chronic lesions—in the pelvic viscera, the lymph glands, the pleura, the peritoneum, in the wall of the newly formed abdominal sac, and fibro-caseous bands. In the viscera this is usually cellular, but may be caseous. In the serous membranes it is always caseous.

(4) Tubercle bacilli are numerous in the tubercular lesions throughout the body. They are especially numerous in the pelvic viscera, lymph glands and abdominal pus sac.

(5) The products of ordinary inflammation are often found associated with the tubercular lesions.

(6) Staphylococci are found in all the organs. They are most numerous in the lining of the abdominal pus sac and the fibro-caseous bands.

(7) Streptococci are likewise found throughout the body, but are especially abundant in the pelvic viscera, the pus sac and the fibro-caseous bands.

(8) Russell's fuchsin bodies are found wherever the tubercular tissue is beginning to degenerate and occasionally where the degeneration is extreme.

(9) The viscera are congested and there is general arteriosclerosis.

CONCLUSIONS.

From the clinical history, gross anatomical lesions and histological appearances, we therefore conclude that the disease began as a tubercular inflammation of low grade in the pelvic viscera some time before pregnancy.

That the tubercular process spread by direct extension or by the lymph channels to the anterior part of the abdomen, which thus became shut off from the rest of the peritoneal cavity by chronic adhesions.

That the phenomena incident to pregnancy and the puerperium excited the process to increased activity, to which, either at this time or later, was probably superadded a pyogenic infection.

That this infection remained limited to the pelvic organs because of occlusion of the tubes by previous tubercular adhesions or by the more recent inflammatory process, until manual examination of the already much diseased organs caused unavoidable rupture into the anterior abdominal sac.

The pain, rise of temperature and abdominal distension were the result of the secondary pyogenic infection of this cavity, from which the general infection took place.

The general tubercular infection doubtless spread, partly by direct extension, partly by the lymph channels, but principally through the blood-vessels. The first two modes of invasion are shown by the extensive diffuse infiltration of all structures continuous with and contiguous to the pelvic viscera and the abdominal pus sac. Infection through the blood is evidenced by the general miliary tuberculosis of all the viscera of the body.

Tuberculosis of the tubes and peritoneum is usually a secondary condition. It may follow tubercular disease of any organ of the body, but is seen most commonly associated with tuberculosis of the lungs. In this case, however, the process must have originated either in the peritoneum or in the tubes. The lungs showed no old tubercular foci, but were involved only in the general miliary tuberculosis. The pleura was diseased, but to a much less extent than the peritoneum or the tubes. The intestines were not at all involved, and the lymph glands were enlarged and caseous, but not extensively diseased.

The question arises, what was the relation of the tubercular disease in the tubes to that in the peritoneum? Which was primary, which secondary? Considerable difference of opinion exists as to the relation between tubercular peritonitis and tuberculosis of the tubes. In his monograph on "Tubercular Peritonitis" Osler[11] says: "The Fallopian tubes are often affected, but the proportion given by various writers differs much. It is safe to say, I think, that in 30 to 40 per cent. of the cases in women the tubes are found affected. The process is commonly confined to the distal ends and may be primary—which is usual—or is secondary to the peritoneal involvement."

In this opinion of primary tubal involvement the majority of observers agree.

On the other hand Williams[14] asserts that "Tuberculosis of the Fallopian tubes is far more frequently of secondary than of primary origin, and when it occurs in combination with tubercular peritonitis, it is far more often the result of than the cause of the latter. The fact that the fimbriated extremity of the tube is the portion most frequently affected is of itself evidence in favor of its secondary origin, and in several cases of tubercular peritonitis we have found tubercles on the exterior of the tube and its fimbriated end, but none in its interior, showing that the process was extending from above downward." Among others, Sänger and Borschike confirm his view.

In this case the only indication pointing to the peritoneum as the source of disease is the extensive caseous involvement of its anterior part. On the other hand, primary disease of the tubes is indicated by the still more extensive involvement of their walls, by the presence of numerous giant cells, considered by Baumgarten[1] as significant of primary tubal disease, and by the clinical history which points to the pelvic organs as the first affected. As shown by the foregoing studies, the fimbriated extremity of the left Fallopian tube must be regarded as the probable starting point of the whole tubercular process.

If then the tubercular disease of the tube is of primary origin, the further question presents itself, how did the disease arise? Primary tuberculosis of the tubes may originate in two possible ways: (1) By infection through the blood, and (2) by infection from without. Primary infection through the blood is a matter of pure speculation and is highly improbable. The more likely source of infection is through the vagina from without. Although no history of such invasion could

be obtained in this case, yet it is regarded as the most probable portal of entry. The fact that the uterus was less involved than the tube, and that the vagina was not all diseased, does not negative this theory of infection, since the tubercle bacilli, failing to find conditions favorable for their growth until the left fimbriated extremity was reached, here set up their characteristic lesions. Thence they probably spread to the fundus of the uterus, to the uterine cavity and cervix, to the opposite tube, and to the peritoneal cavity, from which the general infection took place.

The associated involvement of pleura and peritoneum must be considered of diagnostic value, since coincident inflammation of both these membranes is so often of tubercular origin.

Besides the extensive character of the lesions, there are several unusual features in the case. In the first place, considerable interest attaches to the fuchsin bodies so numerous in this case. These bodies resemble the little globular masses described by Russell[12] in 1890 as "a characteristic organism of carcinoma," and correspond in every particular with the description of these bodies as given by Lubarsch[9] under the head of "Albuminous Degeneration."

There is scarcely a normal condition or pathological process in which they have not been found, but their relation to malignant tumors has been especially noted. They are not of constant occurrence in any pathological change.

With regard to their nature, the view of Russell that they may be considered as yeast fungi is no longer held. They have been variously interpreted as protozoa, hyaline thrombi, altered Altmann's granules, von Recklinghausen's hyaline, bodies resembling corpora amylacea, transformation of the cell protoplasm, and as a product of the tissue fluids in which a degenerative process is added to a beginning coagulation process.

Lubarsch himself inclines to the view that the Russell's fuchsin bodies are the product of cell protoplasm, the granules of which have undergone certain chemical and physical changes. He believes that they may be due either to a secretion or to a degeneration of the cell, the former occurring in normal conditions, the latter in pathological processes. Lubarsch also refers to the relative increase of basophiles in areas in which the fuchsin bodies abound—a phenomenon noted by Niehus and Klien, and observed also in the present case. In explanation of their coincident occurrence he attributes the formation of the fuchsin bodies to a chemical change and confluence of the granules of the former and some of the wandering cells, and suggests that both are due to active tissue transformation. From the description given in the present case, this mode of origin seems probable.

A second point of interest is the rare combination of tuberculosis in both body and cervix of the uterus. Regarding this Williams[11] says: "The affection is almost always limited to the body of the uterus, rarely extending beyond the os to involve the cervix; and of the few cases of tuberculosis of the cervix on record, a considerable portion occurred without any involvement of the rest of the uterus. Tuberculosis of the uterus which also involves the cervix is rarely met with, and as far as we can ascertain from a careful survey of the literature, has only been noted in seven cases."

In this connection may also be mentioned the tubercular invasion of the muscular coat of the tube, which, as a rule, escapes involvement even in advanced tuberculosis.

The large sacculated exudation occupying the anterior portion of the peritoneal cavity and simulating ovarian cyst is rare. Among the few recorded cases of tubercular peritonitis with encysted collection of fluid are those reported by Bernutz, Spencer Wells, Gardner, Howard, Erich, Ewing, Mears and Atlee.

A feature of special interest is the thick fibro-caseous band which extends across the pus sac in the median line of the abdomen, and is probably the remains of the rolled-up omentum. In structure it bears some resemblance to the thickened strands described by Klebs[8] as passing transversely across the abdominal cavity below the umbilicus, and which he attributed to retraction of the omentum. Such omental tumors are not uncommon and have frequently been described. In location, however, this fibro-caseous band does not correspond with the transverse omental tumors, since its direction is anteroposterior and from above downward, and its attachment is by its extremities. In explanation of this formation one can only assume some distortion of the original omentum, with displacement, thickening, caseation and complete transformation into the fibro-caseous band.

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DESCRIPTION OF PLATE.

Pelvic viscera fixed in formalin and hardened in alcohol, viewed from above. Right tubo-ovarian mass seen in cross-section.

F. U., fundus of uterus.
Right tube, showing cross-sections of tubercular lumen.
r. F. E., fimbriated extremity of right tube.
r. Ov., right ovary.
M., myoma.
Rd. Lig., round ligament (left).
l. T., left tube.
l. F. E., fimbriated extremity of left tube.
l. Ov., left ovary.

From the left surface of the larger myoma projects a fibro-caseous band. To the right is attached a portion of the abdominal pus sac.

A VISIT TO BAD NAUHEIM, WITH THE PURPOSE OF INVESTIGATING THE "SCHOTT TREATMENT" FOR CHRONIC HEART DISEASE.

BY C. N. B. CAMAC, M. D., *First Assistant Resident Physician.*

Last November, at Dr. Osler's suggestion, we undertook to introduce into the hospital the Schott treatment of exercises and medicated baths for cases of chronic heart disease. After consulting the bibliography of the subject, several cases were placed under treatment according to the instructions contained therein. At once, however, we were confronted by numerous questions, answers to which it seemed quite impossible to find in any of the references at hand. Although the literature dealt at length with changes in the cardiac outline, the position of the cardiac maximum impulse and the respiration, the theories upon which the beneficial effects were based, etc., no answers to such practical questions as the following were given:

(1) Is any massage to be employed during or after the bath?

(2) What drugs are to be employed during the treatment, and what drugs are contraindicated?

(3) Should the baths and exercises be given together; or if separately, which should precede?

(4) Are stimulants to be administered before or after the bath?

(5) What should be the diet of the patient?

(6) Are cases of hydrothorax or ascites to be tapped? etc., through quite a list with which it is hardly necessary to weary you.

Finding many of these questions unanswered, it was with considerable interest that I received Dr. Osler's suggestion to visit Bad Nauheim, the home of the treatment and of Dr. Schott, its originator.

Nauheim is in the Grand Duchy of Hesse, three-quarters of an hour from Frankfurt a. M. and two hours from Homburg. Nearly in the centre of the northeastern half of what geologists have called the Mayence Basin (Mainzerbecken) Frankfurt is located, and at the eastern slope of the Johannesburg, the last spur of the Taunus mountains, is situated Bad Nauheim. As one approaches Nauheim he is struck by the great trestle-work structures in the midst of the fields. On examining these more closely they are found to be frame structures about 200 to 300 feet long and about 50 feet high, supporting switches closely stacked one upon another. The salt waters are raised to the top of these trestles and allowed to filter through the interlacing switches, upon which, by the evaporation of the water, the salt is deposited. These switches are removed every few months or so, the salt broken from the branches, ground and refined, and serves as the commercial salt of the surrounding country. The most beautiful forms result from these deposits, and by the clever devices of the natives the most grotesque figures are produced. I have some of the figures thus produced.

An estimation of the commercial value of these works to-day may be made by the value put upon them in 1806, when they were considered by Napoleon an adequate reward to Marshal Louis Nicolas Davout (erroneously written Davoust) for his services in the French army; and again in 1866, when they fell to Hesse Darmstadt in exchange for Homburg. Since 1834 the reputation of Nauheim for the efficacy of its springs has been steadily coming to the notice of Europeans. Frankfurt up to this time forming the centre and battlefield of many of the German disputes with France, rendered Nauheim scarcely a fit place for invalids.

It was therefore not until 1834 that we begin to hear of Nauheim as a resort for invalids. It was not until 1860, however, that Dr. Beneke of Marburg considered scientifically the value of the medicated bath treatment. From 1859–1870 several articles by Beneke of Marburg, upon the waters of Nauheim, appeared in the Berlin. Klin. Woch. From 1870 to 1890 August and Theodore Schott and J. Groedel were frequent contributors on this subject to the Berlin. Klin. Woch., also to the Deutsch. Med. Zeitung. August Schott died, but his brother Theodore continued the work, and published in 1892 an article in the Lancet which caused little comment.

In 1894 W. Bezley Thorn became an ardent advocate of the bath treatment, and published an article in the Lancet and also a small book in which he described quite fully the baths and exercises. With the appearance of this systematic little book up to the present the treatment has been very popular in

England. Nauheim, its waters, and the resistance exercises, have been frequent topics in English and German medical journals. In France and America the treatment has as yet received no very thorough trial. It is interesting to note here the increase in the number of visitors from 1871 to 1895. In 1871 the visitors numbered 5,249; in 1891, 9,244; 1892, 10,272; 1893, 10,384; 1894, 11,681; 1895, 14,136.

Although the season was over when I visited Bad Nauheim, I had the opportunity of seeing the baths through the courtesy of Dr. Hirsch, Dr. Schott's assistant, who showed me over the grounds and described very fully the details of the treatment. It can best be described in Dr. Schott's own words: "The springs of Nauheim may be divided into two classes, those suitable for bathing and those suitable for drinking. Together with other ingredients the bath waters contain from two to three per cent of sodium chloride, from two to three per 1000 of calcium chloride, various salts of iron, above all, very large amounts of carbonic acid.

Coming from the depths of the earth, they have a temperature of 82–95° F. Springing from a depth of 180 metres, supercharged with carbonic acid gas by the pressure to which they are subjected, the waters gush far above the surface; for example, spring No. 12 rises to a height of 56 feet and falls again in white seething masses." This is a most striking condition; so richly charged with carbonic acid are these waters that the reservoir into which they fall has the appearance of a great mass of clouds. "Conveyed directly from the main by means of subterranean pipes, these waters charged with their natural gas are allowed to completely cover the body of the bather. Little bubbles of gas are seen to immediately cover the whole surface of the body; the waters of springs Nos. 7 and 12 escape from a pressure of from 1½ to 2½ atmospheres, and afford a surf bath which compares accurately with the strongest surf bath of sea water."

The first question which arose when this matter came to be scientifically investigated was, how do these baths and exercises act? That they were very efficacious in the relief of chronic cardiac disease had been demonstrated for some years back, but their action had never been investigated. There are several explanations given:

(1) That given by Dr. Schott in the following words: "Physiological research of recent years seems to show that the salts held in solution in water externally applied have no direct action on the system; the light and mobile molecules of the gas, on the other hand, pass rapidly through the skin to the corium with its rich supply of blood. We must look upon the salts held in solution as passing by imbibition through the outermost layer of the epidermis, and so acting on the terminal nerves of the skin as to exert a reflex action on the internal organs. The warm baths act in their own peculiar manner on the organism as a whole; increased tissue change seems to be induced by an increase of the oxygen absorbing power of the cells, and hence follows the sense of the need of rest and sleep as an immediate consequence of the bath, as well as influences speedily brought to bear on the nervous system as a whole. Excessive bathing induces an excitable state of the nervous system, sleeplessness, loss of appetite and consequent loss of strength. The principal changes which ensue in the system and in the function of the special organs are that the heart beats more slowly and strongly, the pulse becomes full and increases in force, and the blood pressure may rise to the extent of 20, 30 mm. of mercury; the breathing becomes regular and quiet, and the capacity of the lungs increased.

While the patient is in the bath he becomes flushed and a feeling of comfort and warmth ensues which may even rise to one of an agreeable intoxicating character. Almost invariably the excretion of urine is increased; exudates in the body cavities, especially from the peritoneum, pericardium and pleura, are absorbed. This latter action and that on the valves of the heart can only be explained on the theory of reflex action produced by influences acting upon the terminal nerves."

Another explanation is that given by Dr. Bezley Thorn, that there is a dilatation of the muscular arteries and afterwards those of the skin, and thus there is a relief of the heart from backward pressure.

In Lauder-Brunton's massage experiments he demonstrates that more blood flows through the massaged part and that blood pressure at first rises and then falls, and that on the conclusion of massage more blood collects in the massaged part. These experiments were confirmed by Dr. Oliver.* T. Grainger Stewart† concludes that the passive exercises (1) improve the circulation of lymph within the tissues, and (2) bring a larger volume of blood into the muscles. He quotes the conclusion of Ludwig to the effect that the capacity of muscles for blood is equal to the combined capacities of the internal organs and the skin. If therefore this be so and Dr. Lauder-Brunton's experiments be correct, the increased amount of blood in the muscles must indicate a relief of the congestion in the internal organs.

In Dr. Schott's explanation there are two actions:

(1) A cutaneous excitation induced by the mineral and gaseous constituents, and

(2) a more prolonged stimulation of the sensory nerves excited by imbibition into the superficial layer of the corium. The salt producing this excitation is the calcium chloride.

Whatever the explanation of their action may be, two points seem established:

(1) That the apex beat alters its position;

(2) the area of cardiac dulness is diminished. These two facts, especially the first one, were most strikingly obvious in our first cases, and both facts were most forcibly demonstrated to me in the cases which I saw abroad. One can scarcely credit the results published until he has seen for himself these marked changes.

The case reported by Dr. Bowles in the Practitioner for July, 1896, shows a change of 3 cm. in the apex beat before and after a bath of ten minutes duration, and he says after his visit to Nauheim, which was made for the purpose of seeing for himself, "that which I thought impossible is shown to be quite possible." This case reported by Dr. Bowles was one of chronic myocarditis, moderate pleural effusion, general anasarca and general enlargement of the heart. The age of

* Brit. Med. Jour., June 13, 1896.

† *Ibid.*, September 19, 1896.

the patient was not given. I shall not at this time attempt to report cases, but merely mention this one of Bowles in order to confirm what has been our experience of the effect of the bath upon *the position of the apex beat*, and many other reports confirmatory of this remarkable change are to be found in the literature on this subject.

The diagrams of the cardiac outline made by Dr. Bowles are not quite accurate, but there can be little difference in opinion as to the position of the maximum cardiac impulse.

To quote Dr. Schott again: "The methods of administering the baths are of the greatest importance. It is advisable to begin with a 1 per cent salt bath containing $\frac{1}{1000}$ of chloride of calcium, freed from gas and at temperatures varying from 92° to 95° F., the bath lasting from six to eight minutes. The course of treatment should be interrupted by frequent intervals of one day. The temperature of the bath should, if possible, be gradually lowered, while the proportion of solids in solution and the duration of the bath are gradually increased. At a later stage it is permissible to proceed to the baths containing carbonic acid. The temperature may then be rapidly lowered, especially if chloride of calcium be added in order to increase the mineral strength of the bath."

The course consists of six baths: the first and the second being simply with salts, calcium chloride and the sodium chloride; the third, fourth, fifth and sixth contain carbonic acid as well as these salts.

The preparation of the baths artificially was taken up especially by W. Bezley Thorn, in London, in 1895, since which time Ewart, Bowles and Broadbent have employed them in London, Moeller in Brussels, and Heinemann in New York. Following the analysis of the Nauheim waters made by the chemist Fresenius of Wiesbaden, the artificial baths may be readily prepared. We have now packages made up at our pharmacy each containing the proportion of salts for the different strengths of the baths, each package corresponding to 40 gallons of water, which is just about enough to entirely immerse the body. The baths of different strengths are given to appropriate cases.

I have not attempted in this note in any way to speak for or against the treatment nor to report cases. I have thought it best for the present simply to give an outline of the trip to Bad Nauheim, the purpose of which was to see the effects of the treatment and to learn something about it with the object of trying it in the Hospital here. We have now five cases under treatment, and I trust by keeping careful records of the effects of these baths and exercises that we shall be able to pass judgment upon the weak points as well as the strong points of the method. Only by a careful trial can one place himself in a position either to recommend or to condemn the treatment. I take this opportunity of expressing my appreciation of the patience with which Dr. Schott heard and answered my many questions. I also wish to thank Dr. Heineman for the instruction in the movements which he so carefully gave me.

In regard to the exercises, which are worthy of a lengthy description, something must be said. They consist of nineteen movements, each movement restrained by the very lightest resistance. This part of the treatment, under the supervision of a physician, is entrusted to the nurses, to whom we have given careful instructions as to the method of carrying it out.

The following are the instructions which we have laid down for the nurses in the administration of the bath, also the chart showing the observation which should be made.*

RULES FOR SCHOTT BATH.

(1) Always understand clearly from the doctor the following points: (1) Strength of the bath to be given; (2) temperature of the bath; (3) length of time patient is to remain in the bath. *Note.*—Give the bath in the morning unless otherwise ordered.

(2) Observe carefully the chart and note the points therein called for. (1) Give bath on an empty stomach. (2) Note the time from the moment patient is immersed to that when he is taken out. (3) Allow the patient to make as little exertion as possible; assist him in every way. (4) A sheet may be drawn over the tub, but not around the patient. (5) Be sure the entire body is immersed. (6) Keep the finger on the pulse during the entire time the patient is in the bath.

Danger Signals.—Cyanosis (bluing of the face), dyspnœa (difficult breathing), apnœa (gasping), inappreciable pulse. On the appearance of any of these, take the patient out of the bath immediately, put him to bed and keep him as quiet as possible. Friction while in the bath is not necessary, but if the fingers and toes become bluish the extremities may be rubbed slightly towards the trunk. Friction should be cautiously employed; when the patient is out of the tub rub him to a glow; give him a glass of milk or cup of bouillon and allow him to rest for an hour.

Diet.—Small quantity q. 4 h. Meat—boiled chicken, mutton chops; eggs, two a day; oysters, raw or panned; vegetables—peas, beans, lettuce; liquids—beef tea, bouillon, cocoa, lemonade, milk. *Note.*—Never give more than 4 ounces of fluid at a time. Should be sipped. Wine—Port, Rhine, sherry, brandy, dram to half ounce.

Note.—Something light (cocoa and toast) should be taken one-half hour before the bath; something light and hot (bouillon, milk punch and toasted crackers) should be taken directly after the bath. If the heart's action is poor, sherry, brandy or port wine may be given after the bath. Last meal to be taken three hours before retiring.

Bath No. I. Sodium chloride, 4 pounds; cal. chlor., 6 ozs.

Bath No. II. Sodium chloride, 5 pounds; cal. chlor., 8 ozs.

Bath No. III. Sodium chloride, 6 pounds; cal. chlor., 10 ounces; sodium bicarb., 6 ounces; HCl, 7 ounces.

Bath No. IV. Sodium chloride, 7 pounds; cal. chlor., 10 ounces; sodium bicarb., 8 ounces; HCl, 12 ounces.

Bath No. V. Sodium chloride, 9 pounds; cal. chlor., 11 ounces; sodium bicarb., 1 pound; HCl, 1 pound.

Bath No. VI. Sodium chloride, 11 pounds; cal. chlor., 12 ounces; sodium bicarb., 1 pound; HCl, 2 pounds.

Each bath consists of 40 gallons of water.

NOTE.—By using a little more $NaHCO_3$ than is required

*These rules are made after perusal of the literature, also from instruction obtained from Dr. Schott personally.

to take up the HCl, the metal tubs may be employed without doing them any harm.

$$\underbrace{NaHCO_3}_{83.75} + \underbrace{HCl}_{36.37} = NaCl + H_2O + CO_2.$$

The Exercises.*

The exercises are called by Dr. Schott "Widerstand-gymnastik," or resistance gymnastics, and consist in slow movements executed by the patient and resisted by the physician or operator. A short interval is allowed after each movement, during which the patient sits down. The exertion employed must be very small, and should cause no increase in respiratory movements, flushing or pallor. The patient should be loosely and lightly clothed, and instructed to breathe quietly. The resistance made should be of such a kind that the patient may always feel himself easily the master. The operator must not grasp or in any way constrict the limb, but should oppose by the hand held flatly. The movements are nineteen in number:

Arm. (1) Arms extended in front of body on a level with shoulder, hands meeting; arms carried out until in line, and brought back to original position. (2) Arms hanging at sides, palms forwards; arms flexed at elbow until tips of fingers touch shoulder, back to original position; *one arm only moved at a time.* (3) Arms down, palms forward, arms carried outwards and upwards until thumbs meet over head; back to original position; one arm only moved at a time. *Not always advisable.* (4) Hands in front of abdomen, fingers flexed so that the second phalanges touch those of opposite hand; arms raised until hands rest on top of head; back to original position. (5) Arms down, palms against thighs, arms raised in parallel planes as high as possible; back to original position.

Trunk. (6) Trunk flexed on hips; return to original position. *Resist with both hands.* (7) Trunk rotated to left, to right; return to original position. *Resist with both hands.* (8) Trunk flexed laterally. *Resist with both hands.* (9) As No. 1, but fists clenched. *Resist with both hands.* (10) As No. 2, but fist clenched. *Resist with both hands.*

Large Arm Movements. (11) Arms down, palms against thighs, *each in turn* raised forwards and upwards until arm is alongside of ear, then turned outward, and arm descends backwards. Not always safe. (12) Arms down, palms to thighs, *both together* moved backwards in parallel planes as far as possible without bending the trunk forwards. Not always safe.

Legs. (13) Thighs in turn flexed on trunk, opposite hand resting on chair. (14) Lower extremities in turn extended fully, and bent on trunk forwards and backwards to extreme limits of movement, opposite hand resting on chair. (15) Legs in turn flexed on thigh, both hands on chair. (16) Feet together, lower extremities in turn abducted as far as possible and brought back to original position, opposite hand on chair.

Hands and Feet. (17) The arms, extended horizontally outwards, are rotated from the shoulder-joint to the extreme limits forwards and backwards. (18) The hands in turn are extended and flexed on the forearm to extreme limits, and brought back in line with arm. *Resist with both hands.* (19) The feet in turn are flexed and extended to extreme limits, and then brought back to their natural position. *Resist with both hands.*

We have arranged these in 5 groups, as in this way they may be more readily committed to memory.

Rules for Operators.

1. Each movement to be performed slowly and evenly at an uniform rate.
2. No movement to be repeated twice in succession in the same limb or group of muscles.
3. Each single or combined movement to be followed by interval of rest. Count five.
4. Patient's breathing should not be accelerated.

I. *Avoid.*
1. Dilatation of the alæ nasi (dilating of nostrils).
2. Drawing of corners of mouth.
3. Duskiness and pallor of cheeks and lips.
4. Yawning.
5. Sweating.
6. Palpitation.

If any of the above make a complete interval, or if excessive, stop the exercises for the day.

5. Direct patient to breathe regularly. If he holds his breath, make him count in a whisper.
6. Do not constrict the part which is being moved.

Johns Hopkins Hospital.

*The description of each movement is taken (with a few modifications) from "Chronic Disease of the Heart" by W. Bezley Thorne.

SCHOTT METHOD OF BATHS AND RESISTED MOVEMENTS.

Diagnosis..... *Name*..... *Hospital No.*..... *Ward*.....

Date	Bath and Temp. of same and Movements	TIME		Respiration, Pulse and Heart [Pulse Tracing / Cardiac Outline] SEE HISTORY (Should be recorded once a week)												Pulse Pressure		Calibre of Radial		Point Maximum Impulse.			Color of Skin {White / Livid / Cyanosed / Red}			Amt. Urine in 24 hrs.	Remarks
				In bed		Just before		1st half		2nd half		Immed. after		10 min. after													
		Begin	End	R.	P.	R.	P.	R.	P.	R.	P.	R.	P.	R.	P.	Before	After	Before	After	Before	During	After	Before	During	After		

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A CASE OF POROKERATOSIS (MIBELLI) OR HYPERKERATOSIS EXCENTRICA (RESPIGHI) WITH A REMARKABLE FAMILY HISTORY.*

PRELIMINARY NOTICE.

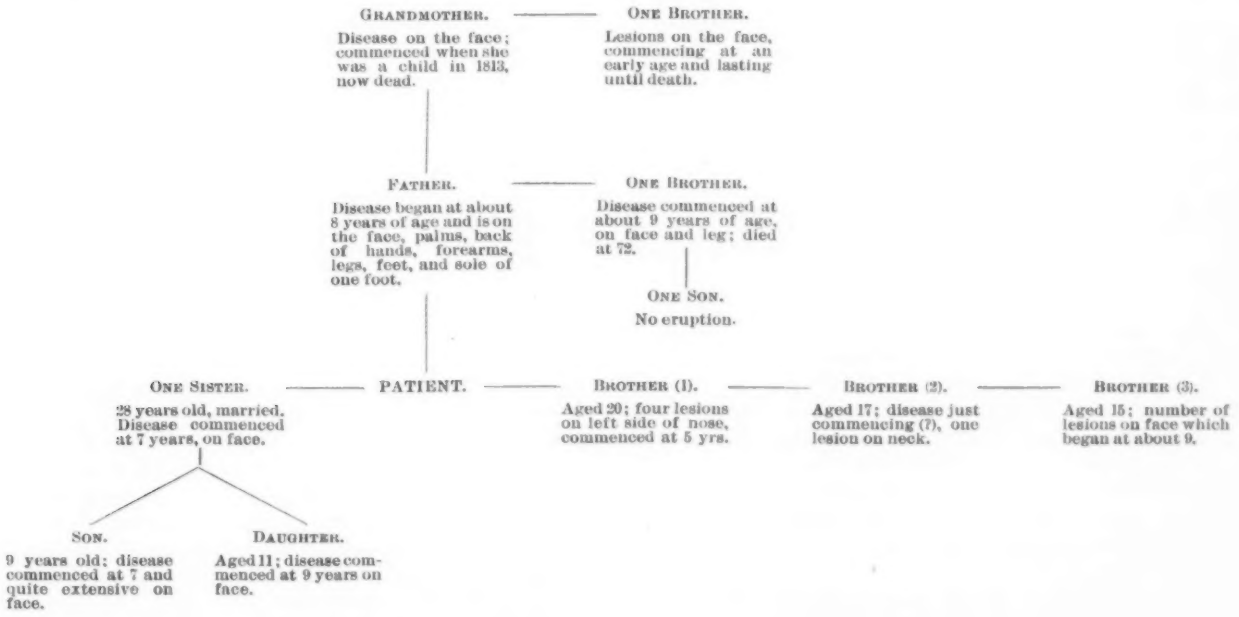
BY T. CASPAR GILCHRIST, M. R. C. S., L. S. A., *Associate in Dermatology, Johns Hopkins University.*

In October, 1893, Mibelli, of Italy, described three (slight references were made to three others) cases of a new disease which, after a careful and detailed histological examination, he named *Porokeratosis*. The disease began at an early age, and was chiefly distributed on the backs of the hands, feet, other portions of the extremities, neck, face and scalp. The lesions commenced as minute, dirty brown, dry, cone-shaped elevations of different sizes and forms, which became much altered as they very slowly increased in size. The lesions extended centrifugally, and the central portion gradually sank in and still remained callous, but the margin was represented by a raised wavy ridge which presented the features of a raised seam. Some patches grew to a very large size and covered almost the whole forearm. Mibelli gave the name porokeratosis to this disease because the most important anatomical lesion was the hyperkeratosis of the sweat duct and sweat pore. In one instance a brother and sister had the disease, mainly on the face and neck. The disease was exceedingly chronic and was unaccompanied by any inflammation or any subjective symptoms.

On January 15, 1894, Respighi, also of Italy, described seven cases of a disease which was recognized by Mibelli as belonging to the same group. Respighi gave the name of "Hyperkeratosis excentrica" to this affection. In one of the cases the father of the patient also was affected with similar lesions. Two years later, three more cases were described by the same author. A single case has been recorded outside of Italy, by Dr. M. B. Hutchins, of Atlanta, Ga., in the *Journal of Cutaneous and Genito-Urinary Diseases* for October, 1896. In Hutchins' case the disease began at two years of age, on the palm of the left hand. The patient is now a man, 32 years old, and has the disease on the left palm, the back of the hand and forearm, and on the face. No histological examination was made. Hutchins compares the boundary of the patches to the "outside of a seam with a thread-like line dividing its lateral halves, and consisting of horny epidermis." No other member of the family had it. In all the cases which have thus far been recorded, only two gave histories of other members of the same family having the disease. In the one case (Mibelli's) a brother had it, and in another (Respighi's), the father of the patient had it.

The patient whom I exhibit this evening is a young man, 21 years of age, who has had the disease since he was five years old, when it commenced on his ears, nose, chin, neck, back of hands and right forearm. The lesions were extremely slow in appearing and in growing. The most remarkable feature about the whole case is the family history, which is here given in a tabular form, together with a very brief description of the disease in each case.

*Exhibited before the Johns Hopkins Hospital Medical Society, March 1, 1897.


GRANDMOTHER.
Disease on the face; commenced when she was a child in 1813, now dead.
ONE BROTHER.
Lesions on the face, commencing at an early age and lasting until death.
FATHER.
Disease began at about 8 years of age and is on the face, palms, back of hands, forearms, legs, feet, and sole of one foot.
ONE BROTHER.
Disease commenced at about 9 years of age, on face and leg; died at 72.
ONE SON.
No eruption.
ONE SISTER.
28 years old, married. Disease commenced at 7 years, on face.
PATIENT.
BROTHER (1).
Aged 20; four lesions on left side of nose, commenced at 5 yrs.
BROTHER (2).
Aged 17; disease just commencing (?), one lesion on neck.
BROTHER (3).
Aged 15; number of lesions on face which began at about 9.
SON.
9 years old; disease commenced at 7 and quite extensive on face.
DAUGHTER.
Aged 11; disease commenced at 9 years on face.

It will be seen that eleven persons in one family have had this disease. The patient gave me this history after careful inquiry, and I have since been able to verify his statements with reference to seven of the cases, viz. father, three brothers, a married sister and two of her children, by personal observation. The patient's description of the lesions in his father and brothers, whom I examined later, was so correct that I feel confident the other descriptions are also true, especially since the remaining family history was verified by his father and mother. The father of the patient also described to me the disease as it existed on his mother, an uncle and brother. I will not go any further into the history or description of the lesions occurring in other members of the family, but will reserve that for a more detailed histological description with photograph and drawings, which will appear later.

The patient first came under my care eighteen months ago, and my attention was then only directed to the lesions on the face. I did not diagnose the disease until I had made ten or more histological examinations of excised portions and had seen Mibelli's and Respighi's articles.

The eruption consists of lesions of various sizes and forms which appear to take on two characters according to their age. The smallest variety, which are distributed chiefly over the face, consist of minute (less than 1 mm. in diameter), dirty brown, semi-globular elevations of a horny nature. When they reach the size of a small pinhead the centre becomes depressed and the margin in some is slightly raised, round, oval or slightly irregular, and presents the appearance of a raised seam, along the centre of which runs a thin black line. As the patch becomes very slowly larger the base takes on a somewhat atrophic character. In a few of the lesions, especially those on the neck, a number of minute conical elevations are distributed along the ridge, giving it an irregular appearance, and sometimes one or two of these minute cones appear in the central portion of the plaque. The largest patches are about the size of a split pea.

After removing some of the diseased portions with a curette and applying very thoroughly the silver nitrate stick, I have seen on four or five occasions the disease return within a month or two in the manner described, viz. a very minute, dirty brown papule, which within two or three weeks apparently began to clear up in the centre. Other lesions return in the form of a ring or oval ridge. If one of the patches is curetted it is fairly easily removed, but the operation is followed by almost as much bleeding as the removal of a small epithelioma. Examination of the scrapings, either fresh or after treatment with liquor potassæ, is negative. While watching the course of the disease week by week, I have seen new lesions arise which I had not detected the week previous.

One or two of the lesions appeared to have formed round a hair follicle, but others did not exhibit any such relationship.

A histological examination of a large number of sections from the most recent as well as from the oldest patches showed that the disease consisted of a marked hyperkeratosis of the sweat pore and duct and of the adjacent hair follicle. In some of the material excised from the face it was not clear that the hyperkeratosis had commenced in the mouth of a hair follicle, but in other sections, especially of the smallest variety of lesions, the disease had undoubtedly commenced about the sweat pore. The oldest lesion, especially from the ear, presented a picture almost identical with that of a mild psorospermosis follicularis (Darier).

From the clinical and histological characters of the disease there was no doubt of the diagnosis of all the cases, but the special feature of this rare lesion was perhaps most marked in the case of the father of the patient, who presented on the hands lesions which showed the well-defined raised wavy edge with a thin blackish line along the centre.

The character of the disease on the hands agreed perfectly with the descriptions of Mibelli and Hutchins. Mibelli has reported 6 cases, Respighi 10 cases, and Hutchins 1. I am able to record a group of eleven in one family, which fact seems at first sight to point to a strong hereditary taint. The histological features will be discussed in detail and a fuller clinical description will be given in a later article.

A RAPID METHOD OF MAKING PERMANENT SPECIMENS FROM FROZEN SECTIONS BY THE USE OF FORMALIN.

By Thomas S. Cullen, M. B., *Resident Gynecologist.*

In April, 1895, I published two methods under the above title in the Bulletin.

Since then, numerous requests have been made for reprints or for copies of the Bulletin of that number, and as the supply is exhausted, it has been thought best to publish the article again with one or two minor alterations. The methods have been continuously employed in the Hospital, and especially in the gynecological department, and have proved uniformly satisfactory.

A complete freezing outfit has been placed in close proximity to the operating room, so that as little delay as possible may occur in examining a specimen. For example, if a carcinoma of the uterus is suspected, the patient is brought to the operating room prepared for hysterectomy. The uterus is curetted and the scrapings are examined while the usual preparations for abdominal section are being made. By the time all preparations are completed the diagnosis is given; if negative, the patient is returned to the ward with the assurance that there is no cause for alarm; if positive, the organ is immediately removed. The woman is thus saved from taking an anæsthetic twice, and avoids the period of anxious suspense of four or five days generally required by the ordinary methods to ascertain whether she has malignant trouble or not.

Any one who has hardened tissues in formalin will be im-

pressed with the rapidity of its action, with the firm consistence of the tissue, and with the absence of the contraction of the specimen so often seen when alcohol is used as the hardening medium. Microscopical examination of a specimen hardened in formalin, as we all know, shows almost perfect preservation of the cellular structure. Recently it occurred to me that formalin might be used in the preparation of frozen sections.

One of the greatest difficulties experienced in rendering frozen sections permanent lies in the fact that when passed through alcohol the section frequently not only contracts but contracts irregularly, distorting the specimen; further, such specimens will often stain imperfectly. The use of formalin will obviate these difficulties, allowing one to make an excellent permanent specimen from the frozen section. My method is as follows: The tissue to be examined is frozen with carbonic acid or ether and then cut; the sections are then placed in 5 per cent. watery solution of formalin for 3 to 5 minutes, or longer if desired; in 50 per cent. alcohol 3 minutes, and in absolute alcohol 1 minute. The tissue is now thoroughly hardened and can be treated as an ordinary celloidin section, being stained and mounted in the usual way. On examining this mounted section one might readily take it for a well preserved alcoholic specimen. Supposing we stain with hæmatoxylin and eosin, the entire process is as follows:

Method I. *a.* Place the frozen section in 5 per cent. aq. sol. formalin for 3 to 5 minutes.

b. Leave in 50 per cent. alcohol 3 minutes.*

c. In absolute alcohol 1 minute.

d. Wash out in water.

e. Stain in hæmatoxylin for 2 minutes.

f. Decolorize in acid alcohol.

g. Rinse in water.

h. Stain with eosin.

i. Transfer to 95 per cent. alcohol.

j. Pass through absolute alcohol, then through either creasote or oil of cloves, and mount in Canada balsam.

The blood is lost in frozen sections. To overcome this Prof. Welch suggested that the specimen be first fixed in formalin and then frozen. I tried this and found that we were able to preserve the blood, but that it did not stain very distinctly. For convenience this second procedure will be called method II. The essential factor is the same in each case. The latter process, however, requires at least two hours. A small piece of the tissue is thrown into 10 per cent. solution formalin for two or three hours. It is then put on the freezing microtome and thin sections can be readily made. The sections are stained in the usual way. The detailed procedure of method II is as follows:

Method II. *a.* A piece of tissue 1x.5x.2 cm. is placed in 10 per cent. aq. sol. formalin for 2 hours. Rinsed in water.

b. Frozen sections are made.

c. Left in 50 per cent. alcohol 3 minutes.*

d. In absolute alcohol 1 minute.

e. The sections are now run through water and stained in hæmatoxylin for 2 minutes.

f. Decolorized in acid alcohol.

g. Rinsed in water.

h. Stained in eosin.

i. Transferred to 95 per cent. alcohol.

j. Passed through absolute alcohol, then either through creasote or oil of cloves, and mounted in Canada balsam.

For ordinary use method I is all that is required. Given a piece of tumor from the operating room, it is possible to give as definite a report in 15 minutes as one would be able to give after examining the alcoholic or Müller's fluid specimens at the expiration of two weeks. Method II is of especial value in the examination of uterine scrapings. Instead of putting them in the 95 per cent. alcohol in the operating room, they may be immediately dropped into 10 per cent. aq. sol. formalin. By the time the pathologist receives them, which is at least two hours afterwards, they are firm enough to be frozen without difficulty, and permanent sections can be immediately made. The second method is to be recommended for all delicate tissues. In employing these methods one must remember, as for example in epithelioma, that some of the cell-nests will drop out, there not being anything to hold them *in situ*, as there is when celloidin is used. We have, however, hardened and stained epithelioma of the cervix by this method without the slightest difficulty.

*The slight modification of Method I, recently suggested by L. Pick, Centralblatt f. Gyn., Bd. XX, S. 1016, 1896, I cannot recommend. When first experimenting with formalin, among other procedures I tried staining the sections after hardening in the formalin and before placing them in alcohol, as Pick now suggests. The results were fair, but the definition so obtained was not to be compared with that gained by first passing through 50 per cent. and absolute alcohol for the short period. I accordingly abandoned it and did not think it worthy of publication.

NOTICE.

All inquiries concerning the admission of free, part pay, or private patients to the Johns Hopkins Hospital should be addressed to Dr. Henry M. Hurd, the Superintendent, at the Hospital.

Letters of inquiry can be sent, which will receive prompt answer, or personal interviews may be held.

Under the directions of the founder of the Hospital the free beds are reserved for the sick poor of Baltimore and its suburbs and for accident cases from Baltimore and the State of Maryland. To other indigent patients a uniform rate of $5.00 per week has been established. The Superintendent has authority to modify these terms to meet the necessity of urgent cases.

The Hospital is designed for cases of acute disease. Cases of chronic disease are not admitted except temporarily. Private patients can be received irrespective of residence. The rates in the private wards are governed by the locality of rooms and range from $20.00 to $35.00 per week. The extras are laundry expenses, massage, the services of an exclusive nurse, the services of a throat, eye, ear and skin or nervous specialist, and surgical fees. Wherever room exists in the private wards and the condition of the patient does not forbid it, companions can be accommodated at the rate of $15.00 per week.

One week's board is payable when a patient is admitted.

DESCRIPTION OF THE JOHNS HOPKINS HOSPITAL.

BY JOHN S. BILLINGS, M. D., LL. D.

Containing 56 large quarto plates, phototypes, and lithographs, with views plans and detail drawings of all the buildings, and their interior arrangements—also wood-cuts of apparatus and fixtures; also 116 pages of letter-press describing the plans followed in the construction, and giving full details of heating-apparatus, ventilation, sewerage and plumbing. Price, bound in cloth, $7.50.

PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS HOSPITAL MEDICAL SOCIETY.

Meeting of December 7, 1896.

DR. THAYER in the Chair.

Typhoid Perforation treated by Surgical Operation.—DR. FINNEY.

In 1762 Richter, in Vienna, first suggested the advisability of opening and draining the peritoneal cavity in cases of general suppurative peritonitis. The operation did not come to general notice, however, until 1884, and was not applied to cases of suspected perforation of the intestine in typhoid fever until 1887. Since that date 46 cases have been reported, to which may be added 6 unpublished cases, including 3 of Dr. Finney's, making a total of 52.

According to statistics taken from various sources, perforation of the intestine occurs in from 1 per cent. to 2 per cent. of all typhoid fever cases. In 80 per cent. of the cases it occurs in a thickened and inflamed Peyer's patch of the ileum, from 2 cm. to 1 m. above the ileo-cæcal valve. In over 12 per cent. it occurs in the large intestine, and in 5 per cent. in the appendix vermiformis. There is usually but one perforation. In twelve cases, however, multiple inflamed Peyer's patches were observed. It occurs more often in men than in women; more frequently between the ages of 20 and 30 years, and usually during the third week of the disease. The duration of life is usually short. In typhoid fever, perforation is always followed by suppurative peritonitis, either general or local; however, peritonitis may arise during the fever without any discoverable perforation. It occurs much more frequently in mild than in severe forms, and the symptoms may be severe, mild, or wanting. The most constant and characteristic symptom is sudden, severe abdominal pain, persisting with increasing intensity; there may be collapse, nausea and vomiting. A few of the cases have shown chill, hiccough, a marked fall of temperature, and absence of the liver dulness. Owing to the great difficulties of diagnosis it is impossible to get data as to the number of recoveries without operation.

A study of the reports of the operations brings out the following facts. The time between the onset of the symptoms of perforation and the operation varied between 2 and 12 hours. The condition of the patients at the time of operation was usually markedly unfavorable. Ether and chloroform were used about equally; in one case both were used. The median incision was the most common. Thirty-three of the cases showed marked general septic peritonitis with foul pus and exudate. Fæcal matter was usually present, and the mesenteric glands opposite the ulcerated Peyer's patches were, as a rule, enlarged and softened. In the treatment, irrigation of the peritoneal cavity was employed more often than simply wiping. As regards the perforation itself, in 8 cases the edges were incised before suturing; in 2 a wedge-shaped piece of the bowel was removed, and in 1 the edges of the bowel were stitched to the abdominal wound while an artificial anus was established. Including all of the 52 cases, there have been 17 recoveries; but if doubtful cases are excluded, the result is 13 recoveries from 47 cases, making a percentage of 27.65. In 19 of the fatal cases autopsies were obtained.

These data are hardly sufficient for general conclusions, but several points are brought out quite prominently in studying the group as a whole. The best time for operation is apparently as soon as possible after the patient has recovered from the shock attending the perforation. This is usually in a few hours. There is a remarkable uniformity in the condition of the peritoneum and viscera; intense congestion, much feculent pus and exudate, with distension of the bowel. As the ileum is the usual place of perforation, it should be examined first; a suture should be taken over any suspicious-looking patches, and the appendix should be removed if it be at all abnormal. If the inflammation does not involve the whole peritoneal surface, irrigation with the necessarily mild fluids might tend to spread the infection. In dealing with the perforation, to excise the edges takes too long, and healing usually takes place without. Should the wall be in such a condition as to make suture impossible, it would be better to pull out the loop of intestine and leave it until the patient had recovered from his fever. The line of suture must be determined by circumstances; the mattress suture of Halsted is to be preferred. Drainage should always be employed. The fact that in 8 cases a wrong diagnosis was made shows how little dependence can be placed on the so-called characteristic symptoms. However, the systematic examination of the blood promises to be of the greatest assistance. During typhoid fever there is no leucocytosis, but immediately after a perforation a marked increase in the number of white corpuscles occurs. Cabot mentions a case of 8,300 before and 24,000 after; in Porter's case the figures are 6,500 and 10,600; in his (Dr. Finney's), 3,000 and 16,400.

Fitz has pointed out the striking similarity of the symptoms of typhoid perforation to those of inflammation of the appendix. Dieulafoy recognizes two forms of typhoid appendicitis capable of producing peritonitis; one, "peritonitis by propagation," involving the lymphoid tissue of the appendix, the other, "para-appendicitis," which is of the usual variety.

The symptoms of peritonitis quickly follow perforation. Examination shows that pyogenic cocci, especially the streptococcus pyogenes, are rarely absent, indicating that they are common inhabitants of the intestine. The Johns Hopkins Hospital autopsy reports show in 4 cases a mixed infection of streptococcus pyogenes and bacillus coli communis, in one a pure culture of the streptococcus, and in one a pure culture of the colon bacillus. The absence of the bacillus of Eberth and Gaffky is probably due to its being destroyed by its more active companions, streptococcus pyogenes and bacillus coli communis. In Dr. Finney's third case pure colon bacillus was found; in other cases the typhoid bacilli were present in liver or spleen or in a peritoneal abscess.

As regards the incision, the median has obvious advantages in general, but if the abdominal muscles are too rigid, it is better made over the part most often affected. Its length should always be amply sufficient. On the other hand, the

time consumed in operation should be as short as possible. The autopsies showed that healing was always well under way. Death usually occurred from septic absorption, but the records show some other complications, obstruction of the intestine, a second perforation, defective drainage or an abscess. General peritonitis makes a case very serious, but with operation the chances are about 1 in 4. The treatment of a case of perforating typhoid ulcer involves three things: (1) Finding and closing the perforation; (2) emptying and cleansing the peritoneal cavity; (3) the establishment and maintenance of effective drainage.

For these ends the following method, employed in his (Dr. Finney's) third case, has proved the most satisfactory. An oblique incision, about 6 inches, is made in the right iliac fossa. The cæcum is found as a guide to the ileum (or appendix); the coils of small intestine, beginning with the ileum, are pulled out systematically. One assistant wipes vigorously and thoroughly the coils with a gauze sponge wrung out of hot salt solution as they are withdrawn, while another keeps them covered with warm sterile gauze. If necessary the entire small intestine is pulled out through the wound and laid to one side, covered with warm gauze. The peritoneal cavity is then wiped out thoroughly, and the coils uncovered and irrigated outside the abdomen with hot salt solution. They are then replaced in the abdomen. The worst or sutured coil is placed with its suture next the abdominal wound, and is packed about with strips of bismuth gauze to insure good drainage. The abdominal wound is closed tightly except a small opening for the gauze drain. In case of distension the bowels should be moved early and thoroughly by calomel in broken doses, followed by salts, and if necessary, a high turpentine and soapsuds enema. If stimulation is necessary, use hypodermics of strychnia, enemata of hot black coffee, or transfusion of a quart or more of the normal salt solution into the cellular tissues under the breast.

Finally, in summing up the experience of himself and others, he concludes that (1) of all the so-called diagnostic signs of perforating typhoid ulcer, most reliance is to be placed upon the development during the third or fourth week of an attack of typhoid fever, of severe, continued abdominal pain, coupled with nausea and vomiting, and at the same time a marked increase in the number of white blood corpuscles.

(2) The surgical is the only rational treatment of perforating typhoid ulcer.

(3) There is no contraindication to the operation, surgically speaking, save a moribund condition of the patient.*

CASE I.—Mr. M., aged about fifty-five years, was taken sick about October 15, 1894, complaining of headache, general malaise, pains in limbs and joints, together with irritability of the bladder. Was seen on October 19 by his physician, Dr. Wesley C. Stick, of Glenville, Pa., to whom I am indebted for the notes of the case. "He gave an indefinite history of having felt badly for about ten days. He had at this time a temperature of 102° F., frequent micturition, bowels constipated, pulse 100. Patient up and dressed; was put to bed and bowels opened by enemata. In a few days temperature came down to about 100° F., pulse to 90. Would not stay in bed. Irritation of bladder ceased. Was getting along very nicely until the night of October 26th, when he was suddenly seized with a violent pain in his abdomen while lying in bed. Was seen the next morning. Pain in abdomen very severe, relieved somewhat by emptying the over-distended bladder through a catheter. He had been unable to void urine during the night, although having frequent and urgent desire to do so. Several hypodermics of morphine were necessary, however, before he became easy. An enema was given with little result. His abdomen was moderately distended, and not very tender to the touch. In the evening of the same day he was seen again and found to have considerable pain, which was relieved by a hypodermic. Had vomited some during the day. His pulse was about 90; temperature 99° to 100° F. The following day, October 28, he vomited frequently a coffee-ground-looking fluid. On the fourth day, October 29, his condition remained about the same; he could get in and out of bed by himself (he was a large-framed, heavy man, weighing about 225 pounds); pulse regular and not above 90; cheerful; the constant regurgitation of coffee-ground fluid being the most troublesome symptom. On the next day, October 30, he was able to sit up in bed, but his pulse had risen to 100 and his temperature to 100° F. Abdomen still not much distended nor very tender."

I saw him at eleven o'clock the night of October 30; his condition then was fairly good, temperature and pulse about 100. Abdomen not much distended, rather retracted; muscles, especially the recti, very rigid; tenderness general, but not marked; frequent and copious vomiting of a dark brownish fluid; countenance pallid and anxious. The diagnosis was general peritonitis, cause unknown, probably appendicitis, although Dr. Stick had from the first suspected typhoid fever with a subsequent perforation of the intestine. The patient had been exposed to typhoid contagion during a recent visit.

As his condition was evidently becoming worse, it was thought best to open the abdomen. The operation was accordingly performed, and under most disadvantageous conditions, about midnight, in a small log house, with very poor light. On opening the peritoneum it was found to be everywhere intensely congested, of a dark chocolate color; the coils of intestine distended and covered here and there with flakes of exudate. There was a considerable quantity of turbid feculent fluid everywhere in the abdomen. A perforation was found in the ileum, about six inches from the cæcum, about one and a half centimetres in diameter, with sloughy edges. These were turned in and sutured with Halsted's mattress suture. The peritoneal cavity was cleansed as thoroughly as possible by sponging, the sutured coil irrigated with salt solution and replaced. The appendix was found normal, save the inflammation of its serous covering. The abdominal cavity was thoroughly drained with strips of iodoform gauze, the ends of which were brought out through the wound. The operation took about one hour. His condition at the end was somewhat collapsed, but he rallied under stimulation, administered hypodermically and by rectum. He began to fail, however, and died about seven hours after the operation. No autopsy.

CASE II.—J. P. D., aged twenty-six years; single; American; tailor; entered the Medical Ward of Johns Hopkins Hospital, August 3, 1895. Family history is negative, except that his father died of some intestinal trouble, the nature of which was unknown, and one brother died, a year ago, of typhoid fever.

Has had diseases of childhood and several attacks of chills and

*Since the above was written one other case has been reported by Armstrong, of Montreal (Montreal Med. Journal, Vol. XXV, No. 8, 1897). This case recovered from an operation for the relief of first perforation. Twenty-four days later, however, a second, and four days later a third perforation occurred, from the effects of which the patient finally succumbed. Reference is also made to the fact that two other fatal cases had occurred in that vicinity.

fever at varying intervals. With these exceptions has been well. Smokes much, does not drink, denies venereal contagion. Present illness began six days before entrance, with severe frontal headaches and a slight chill followed by fever. These symptoms have persisted, together with anorexia; no nose-bleed. For past two days, diarrhœa; has vomited twice; slight abdominal pain, especially before micturition. Temperature on admission, 103.8° F.; pulse, 92. Blood examination negative as to malarial organism; abdomen generally tender, slightly distended; no rose spots; spleen palpable. Leucocytes 3000 per cubic millimetre.

The patient's disease ran a more or less typical severe typhoid course, up to August 16, a. m., thirteen days after entering the hospital, when the following note appeared in the medical history: "Last night patient began to complain of a sharp pain at base of penis, not relieved by catheterization. Temperature has been rather higher, reaching 105° F., pulse 108, regular, though soft and dicrotic. Very nervous; abdomen very tense, though not greatly distended. Everywhere tympanitic; complains of pain; tenderness marked; tongue coated; sweating profusely, and frequently cries out with pain; baths discontinued and cold sponging substituted." P. M. "Patient has been comparatively quiet since a. m., but at about 4 p. m. began to cry out and complain of intense pain in abdomen. Morphine administered hypodermically. Leucocytes, 16,400 per cubic millimetre."

The patient was seen by me with Dr. Thayer at 9.30 p. m., and operation decided upon.

Operation.—Median incision. Peritoneal cavity filled with cloudy, sero-purulent fluid. Peritoneum dark red and much congested. Coils of intestine distended and covered here and there by flakes of lymph. A single perforation, about one centimetre in diameter, was found in the ileum, about sixteen centimetres from ileo-cæcal valve. It was situated, apparently, in the centre of a thickened Peyer's patch, and had sharply punched-out edges. Numbers of raised, red areas could be seen here and there along the lower few feet of the ileum. The loop containing the perforation was drawn out of the abdomen, carefully cleaned of the exuded lymph and fæcal matter, and the rent closed transversely with eight fine, black-silk mattress sutures (Halsted's). Owing to the rigidity of the abdominal walls and the distension of the bowels, puncture of the intestine in several places was necessary, in order to allow of the escape of gas. The loops of intestine were carefully cleansed by irrigation with hot salt solution, and the entire peritoneal cavity thoroughly wiped out with dry gauze. Iodoform gauze was used for drainage, and the abdominal wound closed except where the gauze drainage was brought out.

At end of operation, which lasted a little over an hour, the patient was somewhat collapsed; temperature 103.3° F.; pulse, 150; respiration, 56. He recovered from this somewhat, under vigorous stimulation, and in four hours his temperature was 101.6° F.; pulse, 130; and respiration, 28. His condition did not change materially, except that he became gradually weaker, and died twenty-six hours after the operation. I regret to say that no bacteriological examination was made, and no autopsy allowed.

Case III.—Charles H., German, aged forty-seven years; married; laborer; admitted to Johns Hopkins Hospital, August 15, 1896. Family history good; no cancer or tuberculosis. Has had the usual diseases of childhood, since which time has been strong and well, except an attack of malaria with chills and fevers eight years ago. This attack lasted four weeks. Denies venereal disease; smokes and drinks in moderation. One week before was taken ill with a headache, general malaise, and loss of appetite, and later in the day had a distinct chill, followed by fever. Next day felt somewhat better, but was weak and unable to work. He kept in bed until his admission to the hospital one week later. During this time had had several chills and more or less fever, appetite fairly good, but had eaten nothing but liquids and soft solids. He had no epistaxis nor vomiting; no pain, no diarrhœa, bowels had been regular. At about 8 o'clock on the evening before admission (August 14), while at stool, he was seized with sharp, stabbing pains in right side of abdomen, with marked tenderness to the touch. Nausea and vomiting were present. His physician was called in and gave him morphine hypodermically, which relieved the pain. He passed a fairly comfortable night and was brought to the hospital early the next a. m., when the following condition was noted:

Face pallid and expression anxious; tongue coated, margin and tip clean and red. Temperature, 101° F.; pulse, 116, regular and of good volume. Lungs and heart negative; abdomen is uniformly distended, no undue prominence in any region noted; skin normal; tympanitic throughout; rigidity of abdominal muscles throughout, especially of the right rectus in its lower part. In iliac region indistinct tumefaction felt; tenderness general, but more marked over right lower quadrant. Rectal examination negative; no eruption on skin; urine showed nothing abnormal.

A diagnosis of beginning general peritonitis, probably due to appendicitis, was made. I confess, at the time, the possibility of perforating typhoid ulcer did not occur to me.

Immediate operation advised and agreed to. After the usual preliminary preparation the abdomen was opened through an incision along outer border of right rectus. Abdominal wall distinctly œdematous. The peritoneum was everywhere intensely congested, roughened and dull, with flakes of plastic lymph. In the pockets between coils of intestine and extending down into the pelvis there was an accumulation of thin, yellowish, turbid fluid, containing flakes of exudate. There was no marked fæcal odor to this fluid. The intestines were distended, congested, and dark colored. In passing towards the median line and hypogastric region the congestion and peritonitis became less marked, and in the upper left quadrant the peritoneum seemed fairly normal. At no place was there the slightest attempt apparent at walling off the inflammatory process. The appendix was found in its normal position, somewhat thickened and congested, and constricted at about its centre, due to old adhesions. It was ligated, excised, and stump covered with peritoneal cuff. About fourteen inches from ileo-cæcal valve, in the ileum on its free border, a small hole, about four millimetres in diameter, was found. It was sharply defined, edges bright red and thickened. It was in the middle of what appeared to be a swollen and enlarged Peyer's patch. Soft yellow fæcal matter was exuding from the opening. Opposite the ulcer was a mesenteric gland enlarged to the size of an olive, soft and of a bright red color. The loop of ileum was brought out through the wound and packed about with gauze. The edges of the ulcer were turned in and sutured with eight mattress sutures of fine black silk. The edges of the ulcer were not excised. After suturing, the loop was thoroughly cleansed with salt solution and dry gauze and returned, the peritoneal cavity thoroughly and systematically wiped out with pledgets of dry gauze. Some distance below in the ileum a second swollen Peyer's patch could be made out. Bismuth gauze in strips was packed loosely about the sutured portion, and the ends brought out through the lower end of the incision. The rest of it was closed with buried sutures of silver wire. The wound was dressed with silver foil and sterile gauze. His condition at the end of the operation was good. Time of operation one and a half hours. Pulse, 120, and of good volume; respiration, 20 to 25, regular; color good; no sweating or coldness of extremities. Shortly after operation an enema of several ounces of black coffee and peptonized milk given.

August 16. Patient passed a fairly comfortable night. Pain not so severe as before operation; very little distension; says he feels better. Temperature and pulse still somewhat elevated, 102° F. and 110 respectively. His general condition improved steadily for about a week, when his temperature and pulse began to go up a little. His tongue became more coated, but abdomen not distended nor tender. Spleen not palpable; no rose spots. An offensive discharge

of pus from the right ear indicated an otitis media. About this time a severe pain in the lower part of left thorax developed. Auscultation detected a slight friction rub beginning just below the nipple and running into axillary line.

Blood cultures aspirated from spleen were negative. His temperature and pulse almost reached the normal on August 28, and remained there for a week, when a sharp rise, accompanied by a chill, occurred. His second relapse was quite a severe and prolonged one, so much so that he was transferred to Dr. Osler's ward, where he could be given the cold-bath treatment more conveniently. On September 14 the following note was made by Dr. Osler: "Tongue a little dry; temperature has been continuously elevated since September 5. Between 6th and 9th not above 100° F. Since 9th above 101° F. On the 12th and 13th, 104°, and on 14th, 105° F.; pulse, 128. Complains of pain in abdomen, which is flat and natural looking. Respiratory movements well marked; no tension; everywhere soft. Pain referred to hypogastric region and about navel. Edge of spleen not palpable." During the ten days, from September 17 to 27, his temperature ranged about 103° F., occasionally going above 104° F. He was given thirty-eight tub-baths during this period. It reached the normal on October 6, and remained there.

During this time he developed great tenderness in the toes of both feet, and extending up the front of right leg to the knee was some redness and stiffness. This gradually subsided. Several small furuncles on buttocks and sacrum appeared, which were opened. On October 23 he was well enough to be up for the first time. Improved steadily up to November 2, when a phlebitis of right femoral vein with pain and swelling of leg developed. This gradually subsided, and he eventually made a perfect recovery.

Cultures taken at the time of operation from the peritoneal cavity and edges of ulcer showed pure culture of bacillus coli communis.

Dr. OSLER.—It may interest you to know of the number of cases of perforation in our series. Since the hospital opened we have had about 530 cases (I have not the exact figures for this year) of typhoid fever, with 48 deaths, of which 16 occurred from perforation. The percentage of death from this cause is unusually high, for I think Dr. Finney mentioned it was the cause of death only in about six per cent. of the fatal cases.

That two of Dr. Finney's cases were supposed to be appendicitis is a point of great interest. It is not the first nor will it be the last time that this mistake has been made. There are instances in which patients with typhoid fever without perforation have been operated upon for appendicitis with a fever of five or six days' duration and a swelling in the right iliac fossa. Patient has been admitted to the hospital, operated upon for appendicitis, no perforation found, but an enlarged ileum and swollen mesenteric glands. It is to be borne in mind, however, how frequent is perforation of the appendix among cases of perforation in typhoid. We have had at least four or five in our cases. There have been two cases of recovery in which symptoms of appendicitis in typhoid seemed very clear.

One word as to the time of the operation. This case was unusually favorable inasmuch as it was seen early and the perforation occurred before the 8th day, which is, perhaps, as soon as ever it occurs in typhoid fever. I think the very early and the very late cases will probably be the most favorable for operation. The statistics that Dr. Finney gives are certainly most encouraging.

NOTES ON NEW BOOKS.

Transactions of the American Pediatric Society, held at Virginia Hot Springs, May 27, 28, 29, 1895. Edited by FLOYD M. CRANDALL. (Philadelphia, 1896. Reprinted from the Archives of Pediatrics.)

The meetings of this society are characterized by the practical papers presented and the general discussion of them by the members present.

The present report of its seventh session contains a number of articles relative to the more important diseases of infancy and childhood, with discussion of many of the newer remedies. The list of contributors includes many of the best-known writers on the subjects mentioned.

The treatment of diphtheria with antitoxin as an immunizing agent, and its toxic effects, are especially dwelt upon. A resolution was finally adopted that in the opinion of the society the evidences thus far produced of the beneficial action of this remedy justify its further and extensive trial.

Several papers on anomalous forms of scarlet fever and eruptions simulating this disease, and one on the use of icthyol ointment for the local treatment of the eruption of scarlet fever, are worthy of special notice.

With regard to the use of the antitoxin of tetanus in tetanus of the newborn, the evidence seems to show that up to this time it is uncertain in its action, and that chloral hydrate with one of the bromides is to be preferred.

The antitoxin of erysipelas seems to have had no influence in treatment of sarcoma of the kidney, but the author of the paper is inclined to the view that any foreign substance injected into or near the substance of a sarcoma tends to effect a cure, by setting up degenerative changes in the neoplasm, hence the apparent favorable action of erysipelas antitoxin in sarcomata which are external and accessible, and its failure in visceral sarcomata.

There are also papers on purulent otitis media, "inanition" fever in the newly born, the frequency of typhoid fever in children under three or four years, hyperpyrexia, pygopagus, the neuroses of childhood, tetany, symmetrical gangrene following scarlet fever, angina resembling diphtheria with absence of the bacillus, cardiac anomaly, and the association of enormous heart hypertrophy, chronic proliferative peritonitis and recurring ascites with adherent pericardium, in which the view is expressed of the probable progress of the chronic proliferative process along the veins, through the diaphragm, until it involves the peritoneum.

The report is in good type, well edited and arranged.

BOOKS RECEIVED.

A Collection of the Published Writings of William Withey Gull, Bart., M. D., F. R. S. Edited by Theodore Dyke Acland, M. D. Memoir and Addresses. 1896. 8vo. 184 pages. The New Sydenham Society, London.

Proceedings of the American Medico-Psychological Association, at the Fifty-Second Annual Meeting, held in Boston, May 26-29, 1896. 8vo. 332 pages. Published by American Medico-Psychological Association.

Saint Thomas's Hospital Reports. New Series. Edited by Dr. T. D. Acland and Mr. Bernard Pitts. Vol. XXIV. 1897. 8vo. 510+118 pages. J. and A. Churchill, London.

Archives of Clinical Skiagraphy. Edited by Sydney Rowland, B. A. Camb. Vol. I, No. 3, December, 1896. Fol. The Rebman Pub. Co., Limited, London.

Pathological Report Illinois Eastern Hospital for the Insane. 1896. 8vo. 236 pages. The Blakely Printing Co., Chicago.

Twentieth Century Practice. An international encyclopedia of modern medical science by leading authorities of Europe and America. Edited by T. L. Stedman, M. D. Vol. IX. Diseases of the Digestive Organs. 1897. 8vo. 820 pages. William Wood & Co., New York.

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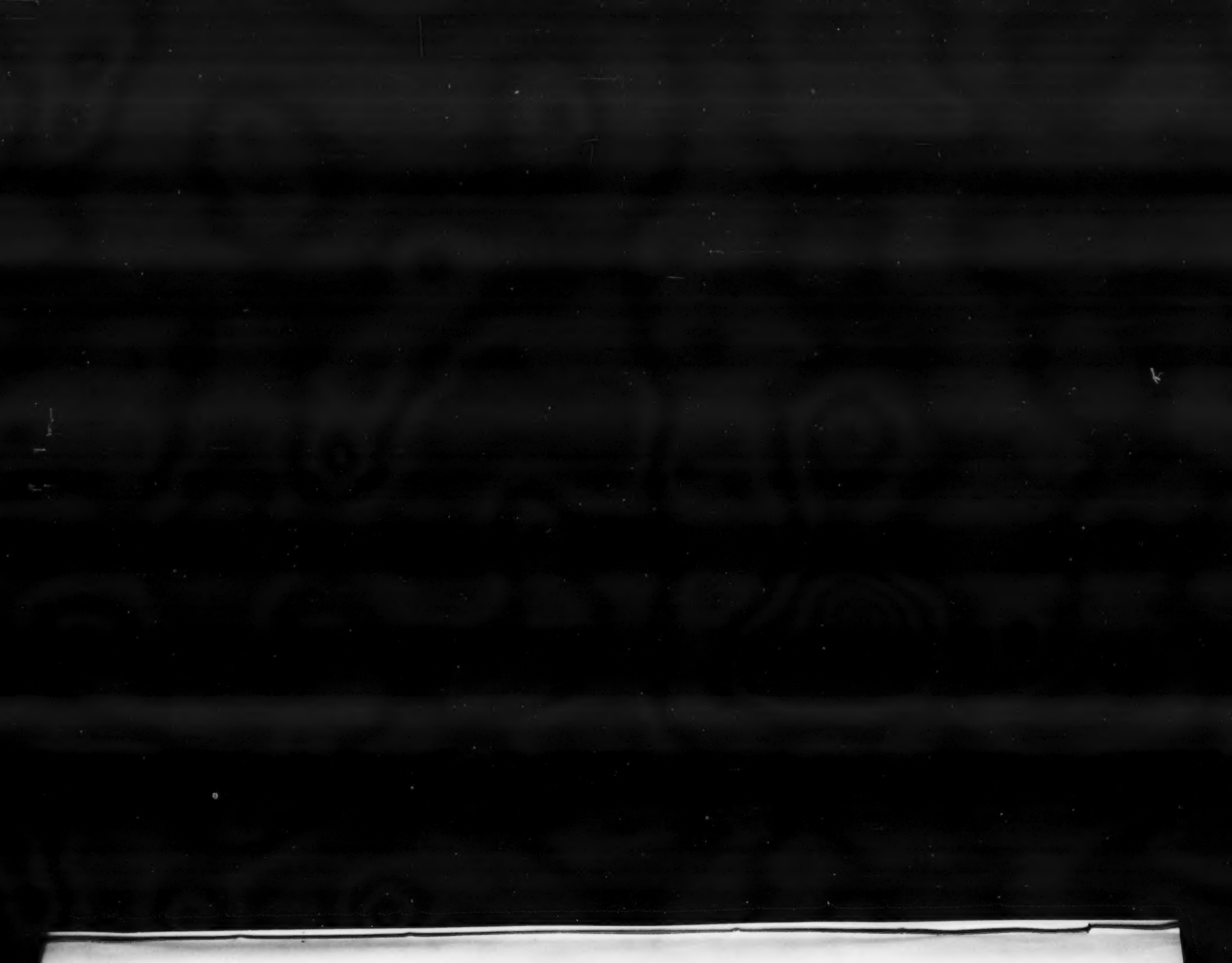
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